Medical Library JAN 13 1925

VOL. LIV JANUARY, 1925 No. 1.

ARCHIVES OF OPHTHALMOLOGY

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ARNOLD KNAPP AND WARD A. HOLDEN

NEW YORK

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REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE DAVID H. WEBSTER

(Complete contents inside)

G. P. PUTNAM'S SONS

2 WEST 45TH ST., NEW YORK

PUBLISHED BIMONTHLY SUBSCRIPTION, SIX DOLLARS A YEAR

Entered at the Post-Office, New Rochelle, N. Y., as Second-Class Mail Matter

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ARCHIVES OF OPHTHALMOLOGY.

A COLLECTIVE STUDY OF THE TREATMENT OF CONGENITAL HYDROPHTHALMUS.[1]

BY DR. EUGENE M. BLAKE, NEW HAVEN, CONN.

THE brief discussion of the treatment of congenital hydrophthalmus, or buphthalmus, in most of the standard works on ophthalmology prompted the writer to attempt to determine from a review of the literature of the past fifteen years and by letters sent to members of this society whether any one form of treatment has been sufficiently successful in the hands of many ophthalmologists to recommend it above others. This seems further desirable since few men have the opportunity to operate upon any large number of cases, large at least as compared with their experience in operating for adult glaucoma. Many of the text books give the student little more information than to say that treatment is disappointing and fail to indicate what, in the author's experience, he considers the best of even the poor measures at hand. Since, however, these cases present themselves for treatment from time to time, the surgeon will wish to know that form of treatment offers his unfortunate patient the best prognosis. This paper, then, will be an attempt to present a cross-section of the accumulated experience of many ophthalmologists concerning the treatment of congenital hydrophthalmus.

[1] Read before American Ophthalmological Society, Hot Springs, Va., June, 1924.

There have been several reports of study of the literature and of questions sent to ophthalmologists regarding buphthalmus. In 1899 Walter Pyle published an exhaustive article and reviewed the various phases of treatment. As Elliott remarks in his book on glaucoma, Pyle's article is especially interesting since it was written before the modern filtering operations were done. His conclusion was that the majority of men were against operative treatment and that miotics alone were of little value, but of the highest service as adjuncts to operative measures. Elliott remarks further "the first thing that strikes one in studying the literature, is that the advocacy of medicinal treatment alone and of the operation of iridectomy has practically dropped out of mention; while references to the newer operations have increased in numbers. Sclerotomy and cyclodialysis also appear to have gone completely out of favor."

In 1913 Dr. Zentmayer reported before the Ophthalmological Section of the A.M.A. the conclusions drawn from questions sent to a considerable number of ophthalmologists. They may be summarized as follows:

Iridectomy was reported to give fair results in 42% and poor in 58%.

Sclerotomy, fair results in 28%, poor in 72%

Sclerectomy, fair results in 40%, encouraging in 20% and poor in 40%.

It is not the intention of this study to go into the interesting question of etiology nor to discuss in detail the histo-pathology of the many eyes which have been studied miscroscopically. A very few facts may be reviewed, however, as an understanding of the development of hydrophthalmus may be of aid in deciding which form of treatment offers the greatest chance of success.

Anatomical investigations by several authors have shown that the foundation of hydrophthalmus is not always the same and that several very different conditions may lead to the classical picture of the disease. All of these causes, however, point to some hindrance to the outflow from the anterior chamber. A few observers still hold to the theory of a hypersecretion of fluid from the inflamed ciliary processes, but many eyes have been examined which were entirely free from all

evidences of inflammation in the uveal tract and which always revealed some developmental defect or complete aplasia of the channels of exit from the anterior chamber.

It may well be that the mechanism underlying hydrophthalmus is an embryological defect and one can do no better than to cite that portion of the description of the disease as it is given in Parson's *Pathology of the Eye.*

"The anterior chamber in fetal life resembles that of lower animals. The ligamentum pectinatum there consists of two parts—an outer more compact part lying in opposition with the sclera and forming the inner wall of the canal of Schlemm, and an inner looser part. The latter persists, in man, until the sixth month, after which it disappears, and with it disappears most of the spaces of Fontana. Rochon-Duvigneaud called the outer persistent part, the sclero-corneal trabecular system, and looked upon it as derived by dissociation from the inner circular lamellæ of the sclerotic. Treacher Collins found that the ligamentum pectinatum in mammals is much more extensive than in man. It consists of an external laminated zone with slit-like spaces, and an inner cavernous zone with large irregular spaces. In man the cavernous zone is practically absent, the angle of the anterior chamber being prolonged farther outward than in mammals' eyes. This alteration is associated with an alteration in the relative size of the cornea and globe. The relative decrease in size of the cornea to the globe in man is thus accompanied by a simplification of the ligamentum pectinatum and a prolongation of the anterior chamber outwards. At one stage in development it passes through a condition exactly like that in mammals. Mr. Treacher Collins concluded from studies that in buphthalmus there is incomplete separation of the iris from the cornea. He is now inclined to feel that the condition is better ascribed to an abnormal persistence of the prenatal condition of the ligamentum pectinatum. This is supported by the fact that in many cases strands of tissue stretch between the base of the iris and the cornea. Collins attributes this to a persistence of part of the cavernous area. He considers that a prolongation of the anterior chamber outward facilitates the exit of aqueous and that this is rendered necessary by the diminished area which the ligamentum pectinatum occupies in proportion to the size of the eyeball. If a

prenatal condition persists in an eye which is congenitally small no delay in the exit of fluid from the eye results and tension remains normal; if, on the other hand, the eyeball attains average size, then delayed exit of fluid occurs and increase of tension with enlargement of the globe results."

In the consideration of the treatment of hydrophthalmus the following measures must be mentioned. (1) Miotics alone; (2) miotics combined with any surgical treatment; (3) iridectomy; (4) anterior sclerotomy; (5) posterior sclerotomy; (6) sclerectomy (La Grange); (7) cyclodialysis; (8) trephine.

I. Miosis alone. The value of miotics is exceedingly difficult to evaluate. Many writers express a firm opinion that it is worthless, others that it is quite as satisfactory as any surgical procedure, and some who laud one type of operation use miotics in conjunction therewith. Certain it is that miotics do not have the effect of opening up the filtration angle, which is partly or completely lacking in 50% of the cases. That it may produce traction on the fibers filling the filtration angle and cause their gradual stretching is not impossible and is the view expressed by Fage and since it does no harm and may be of possible value it would appear to be desirable to make it a routine procedure, trying the effect perhaps for a few months before operation and continuing it after surgical interference.

II. Combined medical and surgical treatment leaves a doubt as to which element has been helpful in the event of any improvement, but is, nevertheless, a precautionary measure. Gadnoski reports from the Heidelberg clinic upon six cases of sclerotomy and miotics combined, with three good results and three bad.

III. *Iridectomy*, being one of the oldest operations for the relief of glaucoma has probably been employed more often in hydrophthalmus than any other surgical measure. It has enthusiastic sponsors and likewise many disasters to its credit. Wolff collected 49 cases operated by iridectomy with the following result: 10 eyes lost through operation, 27 with no improvement and 14 with tension made normal—not a very good showing. Seefelder had among 43 eyes 11 good results, 23 not influenced and 7 made worse (very similar to Wolff's results). In 7 eyes which were operated upon at an early age Seefelder had especially good results and so gives this operation the pre-

ference. Schoenemann obtained good results in 7 patients (13 eyes). He lays stress on the procedure of Schweigger, which makes the incision in the central border of the corneal limbus and also with the avoidance of a peripheral direction of the incision. Reber, in the Würzburger clinic under Hess, reported very unfavorable results from iridectomy. Harlan, Peter and Sydney Stephenson report one successful case each. On the whole, the results of iridectomy are not encouraging. Posey says, "I never remember having had a case of successful iridectomy in buphthalmus."

IV. *Anterior sclerotomy* has been favorably reported upon by numerous writers. Siegrist (in a work by Chaja Spielberg) speaks in favor of it, with or without the combination of an incision of the chamber angle, as done by Vincentiis. It must be frequently repeated, in the hope that a fistulous tract may be formed under the conjunctiva, otherwise, its effect will usually be but transitory. Spielberg, in discussing the pathogenesis of hydrophthalmus, says that he does not think iridectomy will control the tension but that this is better done by an anterior sclerotomy. In this he agrees with Siegrist, Vincentiis, Haab and Stölting. Haab reported 38 cases so treated with 29 good results, 9 lacking in result, and bespeaks a favorable prognosis in cases treated early. Sclerotomy, according to its chief exponents, requires numerous procedures. Thus Haab in 38 eyes of children did 104 operations:

1 sclerotomy in 6 eyes
2 sclerotomies in 13 eyes
3 sclerotomies in 10 eyes
4 sclerotomies in 6 eyes
5 sclerotomies in 1 eye
6 sclerotomies in 1 eye
7 sclerotomies in 1 eye

The treatment averaged 51 days. Stölting had 10 good results in 16 cases and Rochat recommends frequently repeated sclerotomy. Wolff reported 10 cases operated with only 2 good results and Seefelder saw only 3 cures in 13 cases. Priestley Smith had 1 good result in 3 cases.

V. *Posterior sclerotomy* would seem to be only a palliative measure to lower intraocular tension temporarily and cannot

be aimed at the fundamental structural fault in hydrophthalmus.

VI. The *La Grange operation* has not been reported upon by many observers. Fuchs says, "less dangerous and yet successful has proved in my hands an iridectomy in which the incision is very short, but combines with it an exsection of the scleral flap by La Grange's method. Another operation that in hydrophthalmus is less dangerous than iridectomy is trephining by Elliott's method." Allport, Hay and Key have had good results from this form of sclerectomy, especially Key who had six favorable outcomes. Weeks, who favors the La Grange for adult glaucoma, has abandoned its use in favor of the trephine in buphthalmus.

VII. *Cyclodialysis* has been rarely employed. Zeeman reported upon 11 cases so treated in the last ten years with only 3 satisfactory results.

VIII. *Trephine.* One of the main purposes of this investigation of the treatment of hydrophthalmus was to determine whether Elliott's trephine operation, which has been so frequently used for the relief of high intraocular tension, had shown any better results in the congenital type of disease than others. If the results of trephining are discussed more fully than those of other operative procedures it is not because of a bias on the writer's part but in an endeavor to learn whether this newer operation is more dependable than those longer used.

Spital reports upon 12 eyes trephined. In all, 20 trephines were done, 8 eyes once, 2 eyes twice, 1 eye 3 times and 1 eye 5 times. Filtration through the scleral scar could not be proved in any case by the fluorescein method. In ten of these cases iridectomy was combined with the trephine. The results were satisfactory but Spital is inclined to feel that the iridectomy is the important factor. He argues with Fleischer that the scleral opening always closes over because of the great tendency of the young sclera to form scar tissue.

The most complete study of the trephine as applied to hydrophthalmus and its comparison to other operations was that written by Dr. Bruno Fleischer in 1918 from the University Eye Clinic at Tübingen. His results will be largely quoted in this connection.

In the Tübingen clinic the operation is done because of the favorable results which have resulted from the operation from the beginning, which results were confirmed by longer observation. The operation has been performed since 1912 in 16 cases on 23 eyes. Among those are two older individuals and 6 eyes of children which had been operated only one year before and are not included in the consideration. In all cases the results were satisfactory and the enlargement of the eye ceased to progress. In nearly all cases the tension became normal. In the 11 eyes which were followed for periods of 3½ to 6 years a complete healing of the glaucoma resulted.

Fleischer points out that the operation offers no special difficulty. All were trephined above, 3 done twice. A 1.5 or 1.75*mm* trephine was used. Five times a peripheral iridectomy was done and 2 of these required a second operation, indicating that an iridectomy down to the pupillary border is safer. Reposition of the iris is rarely possible and for this reason iridectomy is usually done.

Of the accidents seen, once the iris was only incised, once vitreous prolapsed and once the conjunctival flap was fenestrated. Usually no sutures were used. Chloroform was always employed and a few days preparation with miotics preceded operation. Firm bandaging is important—a dressing, adhesive strips, Fuchs' wire mask and binocular bandage. Atropine was used for a few days until inflammation subsided. On an average, patients were discharged in 20 days. Pilocarpine ½% was used for one year where possible. Retinal detachment was made out twice. A smooth scar results, without fistulazation or bleb formation for the scleral and corneal openings always closed by scar tissue. Marked improvement in behavior and disposition was noted in many children. Intraocular pressure was usually reduced at once but a few cases required considerable time.

"From our material," he says, "we may conclude that the glaucoma of hydrophthalmus is reduced and complete healing with good vision follows if the patients are treated before the end of the first year." After the first year there has usually been so much destruction of the optic nerve that a severe and permanent loss of vision has occurred. The fear that the trephine opening presents a danger in children's eyes has not

been substantiated because the trephine opening is closed by firm scar tisue.

Upon what the favorable effect of trephining in hydrophthalmus depends, is uncertain. The observation in a relatively few cases, that a peripheral iridectomy made necessary a second trephine might lead to the conclusion that the incision down to the pupillary border is the essential moment. This is the view of Seefelder and Schoenemann. Therefore, the favorable action of the trephine with iridectomy as opposed to simple iridectomy would seem to lie in the simpler, less dangerous and less complicated technique. That, however, trephine without iridectomy or with a peripheral iridectomy can lead to healing is proved by a few of his cases.

Bad results from trephine were reported by Bjirke in one case, Gifford in two cases and Casey Wood in one case. Meller and Crigler have each reported one case of late infection following trephining for hydrophthalmus. Gruenert reports four successful cases and Gray Clegg, one. P. H. Adams and Young each report one successful case.

A questionnaire was sent out to 178 members of this society, asking how many cases of buphthalmus they had treated, how many operated upon, and by what methods. It was also asked whether it was felt that miotics are of value and to indicate what method has proved most valuable in their experience in the treatment of this disease. Ninety-six answers were received, some giving valuable information, others stating that their experience was too limited to allow of an expression of an opinion.

The figures given below are, of necessity, not absolute. Where a physician has been in practice for many years and has done much operative work in various public institutions, it could not be expected that he could collect and classify his cases. The results, then, expressed in figures, indicate more than anything the physician's preference in treatment.

The total number of cases treated is 472. Of these it is quite likely that a considerable number are duplications, especially in the larger cities where patients go from clinic to clinic. The answers are, at times, surprising, as some men of wide experience recall but one or two cases treated, while one observer says he has treated "probably 100 cases."

The total number of operations reported is 156, divided as follows: simple iridectomies 78, trephines 50, sclerotomies (chiefly anterior) 15, La Grange 10, iridotasis 3. There are recorded also 10 enucleations done to relieve pain and disfigurement.

Nine of those who answered expressed a preference for iridectomy, 22 considered the trephine the most likely to be helpful, 2 considered La Grange's operation best, 5 sclerotomy and one iridotasis. Twenty-one observers indicated that they felt miotics are of value, and nine felt that miotics are better than any surgical measure. Three members stated a preference for an operation, if miotics failed to reduce tension and the enlargement of the globe, one each for trephine, iridectomy and sclerotomy.

Reviewing the answers to the questionnaire and the literature of the past 15 years, a few conclusions may possibly be drawn. The first and most important is that early treatment is all essential, some measure being employed to reduce intraocular tension before the optic nerve becomes atrophic and further enlargement of the eyeball takes place. This should be done in the first year of life. As to the type of operation which gives best results the facts and preferences would seem to point to the trephine as being most likely to permanently reduce tension. Thus among the members of this society 21 indicated a preference for trephining as against 9 for iridectomy, the older and more widely employed operation.

Probably the principal advantage of the combined trephine and iridectomy is the ease of the procedure under a general anesthetic and the greater safety because of the small opening made into the globe. The larger opening necessary to do a broad iridectomy constitutes a decided menace to the integrity of the globe.

The results of Bruno Fleischer appear to be rather strikingly favorable to the use of the trephine.

From a consideration of the facts presented above it seems to the writer to be the safest plan of procedure to order a miotic, at once, to do a trephine operation before the child is a year old (if the case is seen that early) and to follow operation by the use of miotics for a year. If tension remains high a second or third trephine opening may be necessary to cause an arrest

of the glaucomatous process and check the destruction of the ocular tunics.

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NOCARDIA (STREPTOTHRIX) INFECTIONS.

By Dr. F. E. WOODRUFF, St. Louis.

(With two illustrations on Text-Plate I.)

(1) The most familiar name of this group of microörganisms is streptothrix, but this name had already been used for another genus; therefore, according to the rules of nomenclature, nocardia, which name was proposed by Trevisan in 1889 for the organism discovered by Nocard in farcin des bœuf of cattle, should be employed. Wright calls attention to the misuse of the term streptothrix and gives the reason for the employment of the term nocardia in its place.

From widely scattered localities and at long intervals of time reports have been published describing unique cases of disease produced by varieties of microörganisms belonging to the genus nocardia. In some of these cases points of similarity can be recognized in the clinical symptoms and the gross pathologic lesions, while others differ widely in both respects. They have been found in brain abscess, cerebrospinal meningitis, pneumonic areas and in other pathologic conditions. Eppinger injected cultures into guinea-pigs and rabbits and observed that they caused typical pseudo-tuberculosis. Consolidation of portions of both lungs, thickening of the peritoneum and scattered nodules resembling tubercles were noted by Flexner in a case of human infection as due to nocardia in which the pathologic picture of the disease resembled so nearly that of tuberculosis in human beings that the two diseases could be separated only by finding the causative microörganism in each case. But in no two cases reported up to the present time have the descriptions of the microorganisms found agreed in all particulars. In some cases no

attempt at cultivation was made. In other cases numerous and careful plants on various culture media failed to develop the specific organism. As late as the year 1904 Tuttle was able to find the report of only twelve cases in which nocardia was found in sufficient abundance to have been an important, if not the principal, factor in producing disease. These cases were all fatal, and only once was the character of the disease recognized during life. As the clinical symptoms and the lesions in the human subject as well as in the animals experimentally inoculated with nocardia often resemble those of miliary tuberculosis, the question is naturally suggested whether cases of nocardia tuberculosis are not more numerous than the few reported cases would indicate. The almost universal prevalence of genuine tuberculosis and the extreme gravity of the disease have so long occupied the attention and study of the medical profession that much is taken for granted, and in cases in which the symptoms and lesions resemble with some closeness those characteristic of the well-known disease they may easily be set down without question to the account of the tubercle bacillus. The cases of nocardiosis reported which simulated tuberculosis have been fatal and the lesions for the most part have been widely distributed, but in a number of cases old lesions have been found which suggest that the disease may have been localized for a longer or shorter time, and then, by some accident, may have become rapidly general. In this respect also these cases may resemble tuberculosis. Whether all cases of nocardiosis in the human are general and fatal or, as in tuberculosis and actinomycosis, whether there may be cases of localized disease which recover, are questions which have not yet been decided. The methods employed to demonstrate the presence of tubercle bacilli render nocardia more or less invisible. Again, unless the observer keeps in mind the possibility of nocardia infection, he may not appreciate the importance of finding slender threads with or without branches, and may consider them accidental bacilli, or varieties of leptothrix or non-pathogenic fungi.

As the lungs have appeared to be the seat of the primary infection in most of the cases of human nocardiosis it is very desirable that all cases presenting the physical signs of tuberculosis, in which repeated examinations fail to discover the

ILLUSTRATING DR. F. E. WOODRUFF'S ARTICLE, "NOCARDIA (STREPTOTHRIX) INFECTIONS."

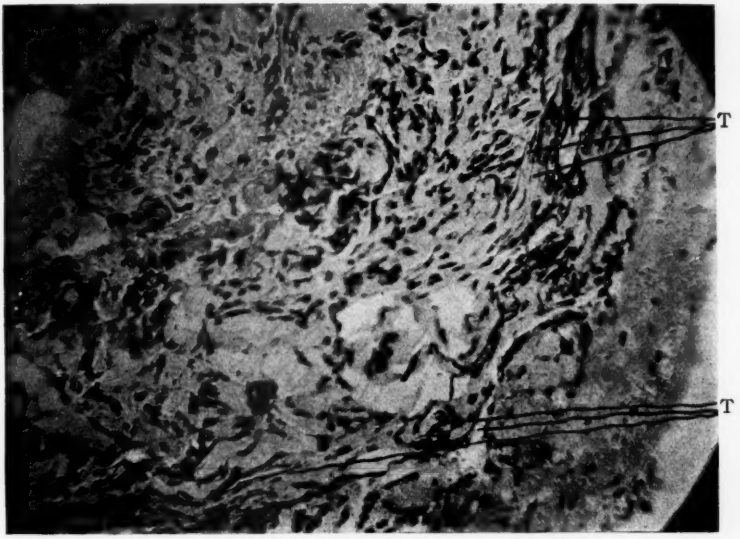

FIG. 1. Section through excised tissue showing thread-like organisms. (T.)

FIG. 2. Thread-like organisms in smears.

tubercle bacillus, should be systematically examined for threads. In this way alone can the frequency of the disease be determined. Varieties of nocardia are widely distributed and are not very infrequently met with, but as yet, with the exceptions mentioned above, very little is known about them.

MICROSCOPIC EXAMINATION.

Smears from the abscess from beneath the skin and on the surface of the kidneys were stained with methyl blue, carbol-fuchsin and by Gram's method. Occasionally irregularly curved, thread-shaped microörganisms are found. They vary considerably in length and thickness and broken and apparently degenerating fragments are seen. The more slender threads are more evenly stained, but some fragmentation or beading of the protoplasm can generally be observed. The thicker threads and broken fragments show deeply stained globules and irregular bodies in a faintly visible rod or thread-shaped covering. Some branching threads are observed, but more commonly they are not branching. Stained by Gram's method, with care not to decolorize too completely, threads like those described in the abscesses are found in great abundance, but rather faintly stained. In the minute foci of small cells one or two fragments of threads are generally seen and a moderate number in the small abscesses. In the areas of more diffuse infiltration these threads are abundant.

CULTURE EXPERIMENTS.

In bouillon the growth is slow. If the tube is not disturbed or jarred, minute white tufts are seen clinging to the surface of the glass. But if the tube is shaken even slightly they sink slowly to the bottom, forming a white, fluffy layer. These growths when undisturbed resemble minute balls of thistle-down. The yellow color is not apparent even in the mass at the bottom of the tube.

MORPHOLOGY.

On blood-serum the threads are comparatively thick and coarse, but those growing in bouillon are very slender and

delicate. The main trunk also is often thicker than the branches. When unstained they are homogeneous gray threads, without any appearance of a central canal or double-contoured wall. There is never any segmentation of the threads. When properly stained there is always a distinct beading or fragmentation of the protoplasm, but overstaining with fuchsin produces rather coarse, evenly red rods. The branching is irregular and without symmetry and the branches are placed at a wide angle, very nearly, and sometimes quite, at right angles. This is best seen in specimens taken from liquid media. The irregularly stellate arrangement of the branches, which was observed by Eppinger in his original specimen, is often seen in young organisms floated out from a liquid medium.

Ferre and Faguet found in Bordeaux, in a cerebral abscess in the centrum ovale, a branching fungus, colored by Gram, which corresponded to nocardia. It grew on agar in round, ochre-colored colonies; on potato there was little growth visible; slimy, tough colonies, which became gray and remained free from white dusting on the surface. Inoculations in rabbits and guinea-pigs were negative.

(2) Pascheff saw a man of 62, who had received a cut on the brow from a shovel twenty-seven years before, which had apparently healed promptly. Thirteen years later, small abscesses appeared which opened and healed. They recurred six years later, with thickening of the brow, proptosis and fistulæ at the external rim of the orbit, which extended to the bone. A deep mass was palpable in the outer part of the orbit. A thread-like organism was obtained on culture and healing was complete after two and one-half months of K. I. treatment. Two years later a new nodule appeared on the temple. This was removed and found to erode the wall of the orbit and extended to the optic foramen. Sections show fibrous tissue with purulent foci containing streptothrix actinomyces. From animal experiments, this was believed to be close to streptothrix farsinici bovis, which has probably never been described in man.

(3) Ginzburg describes one case of actinomyces and one of streptothrix in the lacrimal duct.

(4) The tubercle bacilli not infrequently present projecting

processes or branches, this observation having changed our views regarding the classification of the organism which is probably erroneously placed among the bacilli belong more properly to the higher bacteria.

(5) Ætiology: The nocardia, the infecting microörganisms in nocardiosis have resemblances, on the one hand, to certain bacteria in the structure, thickening and staining of their filaments and, on the other hand, to the hyphomycetes or moulds in their branching, thread-like form and in the production of fine conidia or spore-like reproductive elements out of the substance of the filaments. They differ from the hyphomycetes in that their filaments are more slender and are not tubular structures with double contoured walls, protoplasmic contents and transverse septa. They may be regarded as representing a further transition from the bacteria toward the lower fungi or hyphomycetes than does actinomyces bovis, because they have this property of producing spore-like reproductive elements or conidia, a property not possessed by that microörganism.

Infection seems to take place most frequently through the respiratory tract. In three cases it has occurred from wounds. Malnutrition and chronic disease seems to predispose the individual to this infection as to others.

This idea of the close relationship of the infecting microorganism in these cases to actinomyces bovis is widely accepted, but the writer has shown that it is erroneous and that actinomyces bovis differs so markedly from the microörganisms of this group that it should not be placed in the same genus with them.

It has been pointed out that if the name actinomyces be not used for these microörganisms then the only permissible generic term to apply to them, in accordance with the principles of botanical nomenclature, is nocardia and diseased processes produced by them should be called nocardiosis. In accordance with this view these cases are grouped together under the name nocardiosis and the infecting microörganisms will be called nocardia in this article.

The lesions ascribed to the action of nocardia are of varied character. There is no doubt that these microörganisms produce necrosis, suppuration, abscesses and granulation

tissue formation as well as miliary focal lesions that are essentially suppurative foci surrounded by more or less granulation tissue, but it is not proven that fibrous or calcareous nodules, areas of caseation and tubercle identical in structure with those of tuberculosis, can be produced by them. The best evidence in favor of the idea that lesions, histologically identical with those of tuberculosis, may be produced by them is offered by Flexner; but the microörganism in his case was not studied in cultures and it is therefore not certain that it was nocardia. The filaments of the infecting microörganisms in nocardiosis are scattered among the cells of the lesions or are loosely grouped together and are not aggregated so as to form the compact masses or radiate structures or granules that are characteristic of actinomycosis.

The process in most of the cases has been situated in the lung or has originated there.

Metastatic lesions have been observed in nine cases, among which the brain, myocardium, lung, kidney, peritoneum, lymphatic glands and subcutaneous tissue have been involved.

Fragments of the nocardia may be mistaken for tubercle bacilli, especially for the rare branched forms of that microorganism.

REPORT OF PRESENT CASE.

Lillian I., æt. 19, presented herself on January 6, 1924. Examination showed a round, circumscribed mass about 12*mm* by 4*mm* just under the skin of the left eyebrow at about its middle third. This mass was quite firm and freely movable, being adherent neither to the skin nor to the bony orbit. This was not the first time I had observed this subcutaneous growth in this patient. Five to six years previously she had been observed with the same mass. The mother and patient both thought that this swelling had been first noted by them one year previous to this first visit. There was no history of any fall, blow or injury whatever and the general health had always been good.

Excision of this mass was advised six years ago at the first examination, removal, however, was not done until this time.

The patient stated that in the last few weeks previous to the recent visit this swelling in the brow had become larger. No explanation for this change could be found.

Removal was again advised and done under local anæsthe-

sia. An incision was made along the line of the eyebrow and the mass, which was a trifle below, was pushed up into the field of operation. In dissecting the tumor, the wall was ruptured and a small quantity of pus escaped. The growth, apparently encapsulated, was removed, two silk stitches inserted and the wound closed without a drain. The dressing was changed daily for the next three days at the end of which time union of the skin was quite perfect and dressing was removed. There was however a small amount of soft swelling still present in the region. The patient was not seen again until one week later, when she returned with a localized abscess with fluctuation just below the middle third of the eyebrow. This was incised in the upper part of the eyelid just below the brow and about twelve to fifteen drops of bloody pus allowed to escape. A rubber drain was now inserted and the wound kept open for a few days. After this there was no return of pus formation and now there is only a small amount of scar tissue at the site of each incison.

The excised pieces of tissue from the first operation was placed in 10% Formalin. Smears were made from the discharge at the time of the second operation when only an incision was made. Unfortunately no culture was made, for the nature of the infecting organism was not suspected at that time.

The specimen and smears were given to Dr. H. D. Lamb for pathologic examination. His report follows.

PATHOLOGIC REPORT BY DR. H. D. LAMB.

The piece of excised tissue was about 12*mm* by 4*mm* by 1½*mm*. Macroscopically there was seen scattered over the surface numerous small lobules of fat. After being several days in Formalin the piece of tissue was divided longitudinally to one side of the middle. The surface thus exposed showed quite a dense and firm structure for the mass with fine striations along the middle running apparently longitudinally or in the direction in which the section was made. The color of this cut surface was whitish tinged with gray and showed a long dark streak of hemorrhage also running lengthwise.

Both pieces of the excised mass were de-hydrated, imbedded in paraffin, sectioned and stained with hemotoxylin and eosin.

Microscopic examination of these sections lead to the reconstruction of a mass constituted as follows:

The middle compact portion of the rather flattened elongated piece of tissue is found to be composed of striated voluntary muscle fibers of the occipito-frontalis muscle. All but a few of these fibers run lengthwise of the specimen which explains the fine striations that could be made out macroscopically on the cut surface of the mass. In a few places however muscle fibres are seen to run obliquely and in other places perpendicular to the cut surface. A considerable number of fat cells are seen arranged in very irregular groups about the exterior of the specimen and at one end they are found internally and even in the middle lying between the muscle fibers. Along the sides of the specimen there are found many small lobules composed almost entirely of epithelioid cells although there are seen a small group of striated muscle cross-sections in the midst of several of these lobules. A considerable amount of hemorrhage is diffusely scattered through several of these lobules of epithelioid cells. The nuclei of these epithelioid cells are small, round or oval shaped and stain darkly with hemotoxylin. These nuclei for the most part appear to lie in the center of the meshes of a kind of net work due to a partial disappearance of the cytoplasm of the cells leaving thin dark lines to mark the cell limits. In many places however there are remains of cytoplasm existing in a vacuolated fragmented or granular condition. A moderate amount of rather diffuse infiltration with polymorphonuclear leucocytes is found in these epithelioid lobules. Epithelioid cells are also found occurring singly or in groups of greater or less extent between the muscle fibers, particularly in those portions of the specimen near the two ends. There are present many small veins and capillaries and a few arteries between the muscle fibers throughout the specimen. In the case of several of the capillaries the derivation of epithelioid cells from the endothelial lining can be made out. The essential thread-like organisms are present mostly in compact groups at and near the margins of the lobules of epithelioid cells. They also appear however as small groups in many places between the muscle fibers. The organisms in these sections are mostly short, of varying thickness even in the same thread and sometimes show branching.

The smears from the discharge at the time of the second

operation were stained by Gram's method. They show many long and short threads of quite uniform thickness with some branching. This appearance was rather unlike that of the organisms in the section. It greatly resembles fibrin.

I wish to make acknowledgment to Dr. D. H. Dolley, Professor of Pathology at St. Louis University Medical School who made the diagnosis in this case, also would like to state that these specimens were prepared at the Eye Research Bureau maintained by the Missouri Association for the Blind and the St. Louis University Medical School.

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THE PATHOGENESIS OF GLAUCOMA.[1]

By Dr. F. H. Verhoeff, Boston, Mass.

THE subject that I have been asked to discuss this evening is not of my own selection so that it is unnecessary for me to apologize for the incompleteness of my observations and the lack of finality in my conclusions. While glaucoma is one of the subjects that has been uppermost in my thoughts for many years, and that I have investigated from time to time, my views in regard to it, no doubt like those of most other observers, are still largely in a tentative stage. I shall deal chiefly with primary glaucoma, referring to secondary glaucoma only to the extent I find necessary to establish certain important facts. Secondary glaucoma, however, I believe to be a subject worthy of more extended investigation than it has thus far been accorded.

An apparently great obstacle in the study of the pathogenesis of glaucoma is our lack of quantitative data relating to the activities of living tissues. While physiologists, bio-physicists, and bio-chemists have in recent years contributed much to our knowledge of the behavior of colloids, variations in permeability of cell membranes, the electrical resistance of cells, etc., their observations are not yet sufficiently complete to be of much practical value, especially as regards our understanding of the production and resorption of tissue fluids. Then, too, our ideas regarding such fundamental questions as the source and rate of flow of the aqueous, the normal intraocular pressure and its regulation, have been thrown into con-

[1] Read before the Ophthalmic Section of the Clinical Congress of American College of Surgeons, New York, October 23, 1924.

fusion by recent investigations. Thus, Magitot claims that the aqueous humor is not secreted by the ciliary processes and that it does not flow out through the canal of Schlemm, but under normal conditions it is a practically stagnant fluid. From his writings I am unable to understand his conception of how the normal intraocular pressure is maintained. All the experimental investigations relating to primary glaucoma that have yet been made are, in my opinion, so open to criticism that they are of little if any value.

Yet I do not believe that the question of the pathogenesis of glaucoma is such a hopeless one as may appear from the foregoing remarks. To understand the most important features in the structure of a house it is not necessary to know the minute structure of the bricks that compose it. Similarly, to understand the essential features of glaucoma it may not be necessary to know the details of all the vital processes that are apparently concerned in its causation. In my opinion, if properly interpreted, the clinical, histological, and pathological data which we have collected for years, and especially the results of the experiments which with the aid of nature we have often inadvertently made in our operative work, can lead us far towards the solution of the pathogenesis of glaucoma. My consideration of the subject, therefore, will be chiefly from this point of view.

As regards the pathological histology of glaucoma, I shall confine myself almost entirely to a brief consideration of the changes found at the filtration angle and in the optic disk, because these are of the utmost importance in explaining the elevation of intraocular pressure and the diminution of vision, and because most of the other ocular changes are secondary to them.

In cases of primary glaucoma with elevation of pressure, there is almost constantly found obstruction of the filtration angle by the root of the iris. In the few early cases in which this has not been found it is probable that the root of the iris has separated from the ligament in the preparation of the specimen for examination. In advanced cases the root of the iris is sometimes found free, but the ligament shows sclerosis or other changes which I have elsewhere given reasons for believing are often due to previous adhesion of the iris to the

ligament.[1] After the adhesion has existed for some time the iris root becomes thinner and thinner. Finally its stroma may almost entirely disappear, leaving only the pigment layer. The explanation generally given for this is that the iris is compressed against the ligament, but this explanation is evidently incomplete, since the iris elsewhere is also under the intraocular pressure. If the ligament and cornea formed an impermeable wall the pressure on the two sides of the iris would be the same at the site of the adhesion as elsewhere. As a matter of fact, however, neither of these structures is completely impermeable, and moreover the pressure within them must be less than the intraocular pressure. Hence at the site of the adhesion, fluid must slowly be forced out of the iris and the latter gradually compressed. Fuchs states that the compression of the iris root obstructs the iris vessels, causing atrophy of the remainder of the iris, but it seems to me that there would be no greater pressure upon the vessels here than elsewhere in the iris. In some cases in spite of marked atrophy of the root there is little if any atrophy of the iris proper. Possibly the atrophy of the iris vessels is secondary to the atrophy of the iris rather than the reverse. This is a subject worthy of further investigation.

As a result of modern slit lamp studies, attention has been attracted to the fine pigment found on the back of the cornea in cases of glaucoma. While such pigment is found to some extent in apparently normal eyes of older people, it is apt to be especially abundant in glaucomatous and preglaucomatous eyes. Microscopically I have seldom found it abundant, due, perhaps, to its having been washed off in the preparation of the specimens. It is practically always in single cells which are easily identified as clump cells of the iris derived from the pigment layer. Since an abundant formation of clump cells is by no means restricted to glaucoma, clump cells on the back of the cornea cannot be regarded as characteristic of glaucoma alone. However, in other pathological conditions they are

[1] Sclerosis of the ligament may also occur without obvious cause. I have sometimes found it in cases of secondary glaucoma due to advanced chronic uveitis. Here it may have resulted from the actions of toxins or from the reaction produced by an accumulation of inflammatory cells in the meshes of the ligament.

generally associated with larger precipitates of a different kind. Hence when occurring in large numbers, unaccompanied by other precipitates, they are unquestionably of considerable importance in the early diagnosis of glaucoma. It seems probable that their increased number results from peripheral anterior synechia, but this remains to be demonstrated.

After glaucoma has persisted sufficiently long there occurs cupping of the optic disk. In spite of Schnabel's views it is generally accepted now that this is due to the effect of the intraocular pressure upon the nerve fibers in the disk, and to yielding of the lamina cribrosa. In different eyes the resistance of the lamina no doubt differs greatly so that there is considerable variation in the amount of pressure and length of time required to produce glaucomatous cupping. Associated with the cupping there is disappearance of the nerve fibers in the disk and nerve stem and replacement of them by neuroglia. The latter often, in fact always to some extent, shows cyst-like spaces, sometimes of considerable size. As I have pointed out, such spaces represent simply an exaggeration of the vacuolization which is characteristic of neuroglia in general. Gliomata of the optic nerve often show such spaces to a marked degree, and the neuroglia of a normal nerve shows them on a minute scale. The larger spaces often contain mucin. In fact, it is my belief that with suitable fixation mucin could always be demonstrated within them. For this reason I do not believe that they are directly due to edema resulting from the intraocular pressure, although it is possible that they may be exaggerated by such edema.

In most cases the lamina cribrosa gradually gives way, increasing the injury to the nerve fibers and enlarging the glaucomatous cup. But this does not always happen. Sometimes it recedes slightly if at all, in which case the cup is very shallow. The atrophy of the nerve fibers, however, may be as great as in the usual cases. This raises the question as to whether the atrophy of the nerve fibers is due to pressure upon them in the disk or in the retina. In chronic glaucoma with deep cupping the injury to the fibers evidently takes place in the disk. After an attack of acute glaucoma field changes may be left with slight if any apparent cupping of the disk. This might lead one to suppose that the injury to the nerve fibers had taken

place in the retina, but the fact that the field changes are essentially similar to those of chronic glaucoma impels me to believe that in both conditions the injury to the fibers occurs in the same place, the optic disk. I explain this by assuming that when the intraocular pressure becomes raised the fluid pressure within the retina becomes raised to the same extent so that the retinal nerve fibers are largely protected from injury. In the optic disk the conditions are different, for the fluid pressure within the optic nerve being far lower than the glaucomatous pressure, perhaps even closely approaching that of the sub-vaginal space, permits the nerve fibers to be compressed against the lamina. In other words, in glaucoma fluid is squeezed out of the optic disk as in an opposite sense it is squeezed into it when the intracranial pressure is higher than the intraocular. This explanation evidently applies also to the cases of chronic glaucoma without recession of the lamina cribrosa.

Before going into the question of the cause of primary glaucoma it is necessary to come to some definite conclusion as to the source and outlet of the aqueous humor. This question is no doubt similar to that relating to the cerebro-spinal fluid, but the origin of the latter is also a matter of dispute. The only conceivable sources of the aqueous fluid are the iris, ciliary body, choroid, and retina. In certain pathological conditions under which the pressure in the posterior chamber and vitreous is greatly reduced the anterior chamber undoubtedly may be filled by fluid from the iris. For instance, after scleral puncture for separation of the retina, the anterior chamber may for a time become abnormally deep due to fluid from the iris raising the pressure above that of the vitreous, pushing the iris against the lens and blocking communication between the two chambers. Under normal or elevated pressures, however, it is probable that little of the fluid is derived from the iris, for in cases of complete posterior synechia, or exclusio pupillæ, in which the posterior part of the eye is normal, or relatively so, the iris and lens are pushed forward and remain so after the filtration angle is blocked. This would seem to show that the aqueous is secreted at a higher pressure behind the iris, although an outflow through the iris crypts might possibly explain the difference in pressure. There are, however, excep-

tional cases of secondary glaucoma in which the pressure in the anterior chamber is undoubtedly higher than that in the vitreous. But whether or not aqueous is secreted by the iris under normal conditions, the fact that in cases of congenital or traumatic aniridia the intraocular pressure is normal or elevated shows that the iris is an unimportant source of the aqueous.

The retina can almost be excluded on a priori grounds as an important source of the aqueous, and it is positively excluded as such by the facts that neither complete obstruction of the central artery nor section of the optic nerve and its contained central vessels appreciably lowers the intraocular pressure. Moreover, even complete obstruction of the central vein does not raise the intraocular pressure except in certain cases and in these only after a considerable period of time.

The choroid can be excluded as the source of the aqueous, because if fluid were given off by the choroid it would be at its greatest pressure in the potential sub-retinal space and hence would cause separation of the retina. In cases of sarcoma of the choroid separation of the retina often actually occurs from this cause. The probability, however, is not excluded, that a certain amount of interchange of gases and diffusible substances takes place between the chorio-capillaris and the retina. The function of the great vascular channels in the choroid, in my opinion, is not to regulate the intraocular pressure but probably to serve as a protective cushion for the retina.

By exclusion, therefore, the ciliary body must be the chief source of the aqueous, although a certain amount is no doubt secreted by the iris and retina. The plicated structure and great vascularity of the ciliary processes clearly indicate that they are the part of the ciliary body mainly concerned in the production of this fluid. I may point out in this connection that the capillaries of the processes have the same relation to the ciliary epithelium that the capillaries of the lacrimal and salivary glands have to the gland acini, that is, their walls are closely applied to the cuticular (basement) membrane underlying the epithelium. In the choroid there is also a close relation of the capillaries to the pigment epithelium, but unlike those of the ciliary processes, the capillaries are here separated

from the cuticular membrane by a delicate layer of connective tissue and a fine network of elastic tissue.

The question as to whether or not the ciliary epithelium has a secretory function analogous to that of glandular epithelium I am unable to answer conclusively. Consisting as it does of two layers it would seem that the ciliary epithelium would greatly obstruct the flow of fluid from the ciliary capillaries into the posterior chamber unless it at least was considerably more permeable than ordinary surface epithelium. That it does possess such permeability is proven by the rapidity with which the anterior chamber is reformed and the normal intraocular pressure re-established after paracentesis of the cornea, and that its permeability may vary under certain conditions is suggested by the fact that under inflammatory conditions the intraocular pressure may be abnormally low in spite of marked dilatation of the ciliary vessels.

It is not necessary, however, to assume secretory activity on the part of the ciliary epithelium to explain the maintenance of the normal intraocular pressure or glaucoma. It is simply necessary to assume that the epithelium does not materially reduce the potential pressure of the fluid passing through it, and to assume further that there is sufficient obstruction to the outflow from the eye to permit this potential pressure to manifest itself.

It may be asked why it is if we exclude secretory activity of the ciliary epithelium that fluid is not transuded into the tissue of the ciliary body and sub-choroidal space and the pressure here raised above the intraocular pressure. The answer must be that fluid is transuded here but is carried off and absorbed so much more freely than the aqueous that the pressure remains relatively low. It may be asked also why it is, if we exclude such secretory activity, that the aqueous contains less protein than does tissue lymph. The answer to this is that the intraocular pressure is higher than that within the tissue in general, and hence prevents the transudation of colloids. As a matter of fact, when for any reason the intraocular pressure is abnormally low, the aqueous has a high protein content.

In glaucomatous eyes the amount of wound filtration necessary to reduce the intraocular pressure to normal varies greatly in different cases. In some cases only a barely per-

ceptible amount of drainage is required while in others abundant drainage may be insufficient. This would seem to indicate that in glaucomatous eyes, and probably also in normal eyes, the rate of aqueous secretion, the potential pressure of the secretion, or both, must differ considerably in different cases.

That the filtration angle is an outlet, and, in fact, the chief one for the aqueous, seems to me conclusively demonstrated by the fact that except under special conditions complete obstruction of the filtration angle is always associated with increased intraocular pressure. Then, too, when the structures in the angle are congenitally imperfect, infantile glaucoma results. What other function the spaces of Fontana and the canal of Schlemm could possess it is difficult to imagine.

It has been objected that the venous pressure within the eye must be higher than the intraocular pressure, otherwise the veins would collapse. This is, of course, true, but it is very slightly higher since only slight pressure upon the eye is required to cause collapse of the veins on the optic disk. Now the canal of Schlemm communicates mainly with veins within the sclera and episclera which are necessarily under lower pressure than the uveal and retinal veins, so that the pressure within the canal must be lower than the intraocular pressure. The peculiar structure of the ligamentum, I take it, serves in a most perfect manner to resist the intraocular pressure, thus keeping the lumen of Schlemm's canal widely open and permitting the aqueous humor to drain into it. The spaces of Fontana are not in free communication with each other and the canal of Schlemm, but each seems always to be separated from its neighbor by a thin layer of mesenchyal epithelium (so-called endothelium). This delicate obstruction no doubt prevents too rapid an out-flow from the eye, and it is upon these mesenchymal cells, therefore, that the regulation of the intraocular pressure must largely depend. It seems even possible, although perhaps improbable, that these cells could abstract fluid from the anterior chamber and discharge it into the canal of Schlemm with the pressure in the latter actually higher than the intraocular pressure.

Just how rapid is the outflow of aqueous through the canal of Schlemm, I am unable to say. In enucleated eyes the canal is almost always nearly free from blood, and Troncoso main-

tains that the fact that no dark line can be seen in the filtration angle by any means of illumination proves that the canal of Schlemm contains no blood during life. Since the canal is directly connected with veins, it follows, that if it is free from blood during life there must be a relatively rapid flow of aqueous into it. The rapidity with which a hyphemia disappears also indicates the same thing, since it seems obvious that if red blood corpuscles can readily pass through the ligament, aqueous humor can likewise do so. The canal is so small, however, that a relatively rapid flow of aqueous into it is consistent with a very slight flow through the anterior chamber.

If we assume that the effectiveness of iridectomy in cases of early glaucoma is due to freeing of the filtration angle, it follows that in a normal eye only a small part of the ligament is necessary to keep the intraocular pressure at a normal level.

The conditions that exist in a normal eye, I think, can be best understood by considering what takes place in cases of post-operative separation of the choroid due to wound leakage. In a typical case of this kind the anterior chamber reforms the day after operation. Then the wound gives way at some place, the anterior chamber becomes obliterated and remains so for some time. During this time the intraocular pressure remains almost at zero, due to the free escape of aqueous. The pressure within the choroid now being reduced to the same extent, fluid is transuded from its vessels in greater amounts than can be carried off, distends the sub-choroidal space and causes the choroid slightly to compress the vitreous. Within a few days the wound closes, the intraocular pressure rises and the anterior chamber is reformed. After this happens and after the intraocular pressure has become normal, the separation of the choroid may still persist for a few days or weeks. This proves conclusively that fluid had been squeezed out of the vitreous by the choroid. Gradually the intraocular pressure forces the fluid out of the sub-choroidal space or causes it to become absorbed, thus proving that under normal conditions the intraocular pressure is higher than that in the choroid. It is noteworthy that the retina remains in contact with the choroid throughout, proving that even under conditions of low tension fluid is not necessarily secreted from the choroid towards the vitreous.

Since we know that in practically all, if not all cases of primary glaucoma, peripheral anterior synechia exists, and we know that except in special cases obstruction of the filtration angle always leads to increased intraocular pressure, we must conclude that peripheral anterior synechia is one of the essential causes of primary glaucoma. The most important question connected with the pathogenesis of glaucoma, therefore, is how this adhesion of the iris root is brought about. This question has thus far not been satisfactorily answered.

If we assume that the adhesion antedates the increase in pressure, how are we to explain the shallow anterior chamber? For clinical and histo-pathological observations clearly show that primary obstruction of the filtration angle causes no decrease, but rather an increase in the depth of the anterior chamber.[1]

Thus in ring sarcoma of the iris and in infantile glaucoma the increased pressure is associated with a deep anterior chamber. We must conclude, therefore, that the shallowing of the chamber in primary glaucoma precedes the adhesion of the iris root and also the increased pressure.

As a matter of fact, as pointed out in 1897 by Czermak, clinical observation proves that the shallowing of the anterior chamber always precedes the glaucoma. For it is the rule that in cases of unilateral glaucoma acute or chronic, in which the tension, fields, vision, and optic disk of the unaffected eye are all perfectly normal, the anterior chamber is almost if not quite as shallow as that of the glaucomatous eye. Moreover, in glaucomatous eyes the anterior chamber remains shallow even after the tension has returned to normal either spontaneously or as the result of the use of miotics.

The question at issue really reduces itself, therefore, to that of explaining the shallow anterior chamber of glaucoma. For it is easy to understand that if the lens is pushed forward, the ciliary body will be pulled forward, the ciliary processes pressed against the root of the iris by the zonular fibres, and the filtration angle obliterated.

[1] This fact excludes the recent theory that primary glaucoma is due to the obstruction of the spaces of Fontana by pigment. In the early cases I have examined microscopically, I have not found an important amount of pigment in the ligament.

The theory that the shallow chamber is due solely to increase in size of the lens is untenable, as is also the theory that it is due to congestion of the ciliary body obliterating the circumlental spaces. For primary glaucoma may occur in eyes containing small lenses, and in eyes free from congestion. The theory that increased pressure of the fluid within the vitreous (edema or swelling of the vitreous), pushes the lens forward, as ordinarily understood, is likewise untenable. According to this theory the fluid in the vitreous must obviously come chiefly from some source other than the ciliary body, and as I have pointed out, if the vitreous is otherwise normal, this is impossible. Moreover, it is a simple matter to show that increased pressure of fluid within normal vitreous will not shallow the anterior chamber, at least within a short time. If saline solution is injected into the vitreous of a normal human eye by means of a hypodermic syringe, not the slightest shallowing of the anterior chamber can be detected no matter how great the pressure exerted. This is due to the fact that the outflow of aqueous from the eye is so slow and, as I shall point out presently, the interchange of fluid between the vitreous and aqueous so free, that the increased pressure in the vitreous is immediately transmitted to the aqueous. In some unpublished experiments, I made about ten years ago with Dr. J. M. Love, we maintained a pressure of over 60*mm* of mercury in the vitreous of a living rabbit for six hours without shallowing the anterior chamber. In these experiments there was no canula in the anterior chamber, the outflow from the eye taking place through the natural channels. It is possible, however, that in the case of a human eye the result might have been different.

In connection with the question of the shallowing of the anterior chamber it may be important to know just how much difference in pressure between the aqueous and vitreous is necessary to push the lens forward. So far as I know the only experiment to determine this for a human eye was one carried out by Dr. Love and myself in the series of experiments just referred to. We inserted a large needle through the sclera of a normal human eye (freshly removed for cancer of the eye-lids) and connected it with a pressure bottle. Another needle was inserted through the cornea into the anterior chamber and con-

nected with a different pressure bottle. Starting with a pressure equivalent to 25*mm* of mercury in each chamber, we found that an increase of pressure of 1*mm* in the vitreous would shallow the anterior chamber and an increase of 2*mm* would obliterate it. Judging by this one experiment, therefore, a pressure of 2*mm* of mercury is sufficient to overcome the resistance of the zonule of a normal human eye.

A theory advanced by Curran is that the shallowing of the anterior chamber is due to lack of communication between it and the posterior chamber. His explanation in regard to this I am unable to follow. The only conceivable way, it seems to me, that simple contact of the iris with the lens could shallow the chamber would be by a ball-valve action of the lens. This, however, could produce a difference in pressure between the two chambers equal only to the resultant backward pressure exerted by the iris, which of course is negligible. Moreover, it is excluded as a factor of importance by the fact that generally the anterior chamber remains about as shallow after iridectomy as before.

The theory that distention of the choroidal vessels is the essential cause of primary glaucoma is easily disproved. For while the volume of the choroid could undoubtedly be increased in this way the intraocular pressure would be raised only temporarily if at all, since the pressure would be reduced to normal as soon as an amount of aqueous equal to the increase in volume of the choroid had drained off. Moreover, for reasons already mentioned, if the vitreous was normal the anterior chamber would not be shallowed. The increased intracular pressure could continue only if transudation of fluid took place from the choroid, and this could not occur without causing separation of the retina. Increased vascular pressure in the choroid, if long continued, is no doubt sometimes the cause of secondary glaucoma, for instance in cases of tumors of the choroid, and here separation of the retina does typically occur.[1] In cases of thrombosis of the cavernous sinus there is marked congestion of the choroid, yet there is no glaucoma.

[1] Sarcoma of the choroid, however, can produce glaucoma without separation of the retina. In a case of this kind I found the retina firmly fused with the tumor and the vitreous detached from the retina and pushed far forward.

In a recent case seen by me the tension in each eye was 15*mm* (Schiotz).

Shallowing of the anterior chamber cannot be due to relaxation of the zonule because this would cause tremulousness of the lens, something that does not exist in primary glaucoma. And even if relaxation of the zonule occurred it would still be necessary for the anterior hyaloid membrane to advance, and we know that even after the lens has been removed in capsule this does not occur in non-glaucomatous eyes.

The only possible cause, it seems to me, for the shallowing of the anterior chamber in primary glaucoma is an increase in volume of the vitreous. While much still remains to be learned about the origin and structure of the latter, it has certain features that are sufficiently obvious. It consists of a meshwork of fibrils the finest of which are almost ultramicroscopic in size. The spaces of the meshwork are also minute and are filled by fluid almost if not quite identical with aqueous humor. The actual amount of tissue present in the vitreous is very slight, for if the latter is placed in a gauze bag and its fluid squeezed out, very little substance remains behind.

Towards the surface of the pars optica retinæ the vitreous presents an apparently homogeneous membrane, which may be easily removed from the retina. When deeply stained and examined under high magnification this membrane is seen to give off fine fibrils into the vitreous. That is to say, it seems to consist of fine fibrils so closely compacted as to give it a homogeneous appearance. This membrane thins out and disappears at the ora serrata. Anterior to this, over the posterior half of the ciliary body the vitreous is intimately connected with the elongated unpigmented cells of the ciliary epithelium. Its fine fibrils here seem to fuse with the cell membranes, although it is possible that they form a hyaloid membrane on the surface of the cells so delicate that it cannot be distinguished from the cell membranes. At any rate, traction on the vitreous will not separate it from the cells here. Further forward the fibrils at the periphery of the vitreous gradually become compacted until they form the anterior hyaloid membrane which separates the vitreous from the circumlental space. This membrane continues over the posterior surface of the lens but here becomes less definite. In young individ-

uals the anterior hyaloid membrane is much more obviously composed of compacted fibrils than is the posterior hyaloid membrane, but with increasing age it takes on a more and more homogeneous appearance.

That a relatively free interchange of fluid is possible bebetween the normal vitreous and the posterior chamber is shown by the following facts. In cases of large separations of the retina the volume of the vitreous may be reduced at least one fourth. If a scleral puncture is done the retina may be found replaced and the globe filled out within 24 hours. Later it may again become separated to the same extent within 24 hours. In post-operative separation of the choroid, in many cases more than one fourth of the fluid is expressed from the vitreous and when the wound becomes closed the vitreous may resume its volume in a few days.

From the foregoing facts it is obvious that increase in volume of the vitreous could be produced only by obstruction to the flow of fluid through it. This could be due to decreased permeability of the vitreous tissue, including especially the anterior hyaloid membrane, to change in the character of the fluid, or to both of these conditions.

That the apparent consistency of the vitreous is abnormally great in certain cases is a fact well known to all cataract operators. In most cases of vitreous loss at operation the prolapsed portion quickly collapses and does not interfere with healing of the wound. In some cases, however, it bulges from the wound as a large globular mass, which if excised, is replaced by another. This mass does not disappear until it is constricted off by cicatrization of the wound. Such behavior of the vitreous evidently could be dependent upon the same factors to which are due its increased volume in pre-glaucomatous eyes.

Judging by its microscopic appearance, the anterior hyaloid membrane becomes less permeable with age. The filtration experiments of Nordenson also indicate this, although they probably involved certain sources of error. I have found this membrane especially thick and conspicuous in cases of primary glaucoma, but filtration experiments are needed to prove conclusively that its permeability is diminished in this condition.

Regarding the character of the fluid in the vitreous in pri-

mary glaucoma, the observations hitherto made relate chiefly to its albuminous content. In congestive glaucoma there is certainly often an increase in the protein, but in non-congestive glaucoma, and hence in pre-glaucomatous eyes, there is little if any increase. Increased protein within the vitreous, therefore, is a result rather than a cause of primary glaucoma.

Recently I have made some histological observations that seem to show that the vitreous fluid of glaucomatous eyes, and that of many senile eyes, contains an excess of mucin. The normal vitreous is known to be composed partly of mucin, but this presumably forms part of the tissue, not of the fluid. Mucin is not generally well preserved after ordinary methods of fixation, and in the vitreous especially it is exposed to the action of solvents, during the preparation of specimens. When present it is easily recognized if stained in neutral alum hematoxylin or in thionin. In studying my collection of glaucomatous eyes, I have found mucin[1] abundantly present in the vitreous of a number of them. Since these eyes otherwise appeared similar to those from ordinary cases of primary glaucoma, I assume that my failure to detect mucin in the vitreous of all of them was due to its faulty preservation. I have found also that the cystoid spaces commonly present in senile eyes in the pars optica and pars ciliaris retinæ near the ora serrata so frequently contain mucin that I am convinced that during life they always do so. Such spaces were present in all of the eyes with primary glaucoma that I examined. Frequently I have found mucin on the surface of the posterior part of the pars plana. It seems probable therefore that the mucin in the vitreous is derived from the epithelial cells of the posterior part of the ciliary body and from the anterior part of the pars optica retina.

Presumably the amount of mucin in the vitreous runs parallel with the degree of cystoid degeneration of the peripheral retina, and an excess of mucin in the vitreous is therefore probably a senile condition. Like many other senile conditions it might exceptionally occur relatively early in life, or fail to oc-

[1] The chemistry and physical properties of mucin do not seem to have been thoroughly studied. There are at least three varieties of mucin and in addition a number of mucoids. I have been unable to determine the exact nature of the mucin in the vitreous, but I presume it is epithelial mucin.

cur late in life. Possibly it is dependent upon some general condition which may prove to be controllable.

The theory of glaucoma that I have now arrived at, may be summarized as follows: With increasing age the anterior hyaloid membrane and possibly the whole vitreous body, becomes less permeable, and more and more mucin is secreted into the vitreous. The decreased permeability causes retention of the mucin, which in turn further decreases the permeability of the vitreous, a vicious circle thus being established. The fluid secreted by the posterior part of the ciliary body and retina, being obstructed in its flow through the vitreous, gradually raises the pressure within the latter and increases its volume sufficiently to push the lens forward. This accounts for the more or less shallow anterior chamber in the majority of senile eyes. It is to be noted that this process does not raise the intraocular pressure an appreciable amount but simply creates a slightly higher pressure in the vitreous than in the anterior chamber. In certain eyes the process reaches a stage when the root of the iris is pushed against the ligamentum pectinatum. Finally a sufficiently large part of the filtration angle is obstructed in this way to increase the intraocular pressure. In accordance with differences in the anatomical structure of the eye, this may occur while the anterior chamber is still fairly deep, or may be delayed until the latter is almost obliterated. Smallness of the eye, increased size of the lens, and malposition of the ciliary processes, may be important factors here.

Another factor that may play a part in increasing the pressure within the vitreous is the affinity of colloids for water. If the mucin absorbed water from the aqueous, the pressure in the vitreous would necessarily rise, but how important this factor is remains to be determined.[1]

After glaucoma has become established the depth of the anterior chamber probably depends upon (a) the difference in the rate of absorption of the fluid from the anterior chamber and from the vitreous, (b) the difference between the pressures

[1] This conception calls to mind the theory of Fischer that glaucoma is due to swelling of the colloids of the eye resulting from an acid condition. His theory, however, is based upon experiments so crude as to make it unworthy of serious consideration.

at which the aqueous and vitreous fluids are secreted, (c) the hydrophilic property of the vitreous fluid. These factors could cause the anterior chamber to remain shallow or to become normal in depth. Whether or not in primary glaucoma, the anterior chamber after becoming shallow, sometimes becomes deep spontaneously, I am unable to state from personal observation. It is certainly a fact, however, that in some cases of advanced glaucoma, with complete peripheral anterior synechia, the anterior chamber is almost normal in depth.

To explain the prodromal attacks and temporary exacerbations in glaucoma it is necessary further to elaborate this theory. In chronic glaucoma it seems unlikely that these could be due to temporary changes in the obstruction of the angle, although dilatation of the pupil may play a part here. More likely they are due to normal variations in the secretion of aqueous. That temporary changes which normally occur in the constitution and concentration of the blood, changes in local blood pressure, and changes in the permeability of the capillary walls, must affect the rate and pressure of the aqueous secretion, seems obvious. Under normal conditions of the eye, any increased secretion is compensated for by increased outflow, but when the filtration angle is sufficiently occluded, the increased secretion must raise the intraocular pressure.

In the case of acute glaucoma the problem seems still more difficult. Here the intraocular pressure may rise within a short period of time from low normal to 60*mm* Hg or more. Moreover, in some cases it may spontaneously recede to normal. There is no question but that emotional stress can precipitate such an attack in a preglaucomatous eye, but just how it does so is difficult to determine. Dilatation of the pupil, alteration in capillary pressure, distention of the choroidal vessels, changes in permeability of the capillary walls brought about through vaso-motor nerve disturbance, may be the explanation, or it may be that a rapid change is brought about in the blood through the activity of the endocrine system. As the intraocular pressure rises the intraocular veins are compressed and a vicious circle probably established. As to how this is broken I am not prepared to offer a satisfactory explanation.

The evidence, especially the experimental evidence, regard-

ing the effect of the cervical sympathetic nerve upon the vascular tissues of the eye and upon the intraocular pressure is so conflicting that it is difficult to interpret. On the theory, generally held, that this nerve has a vaso-constrictor function we should expect that irritation of it would reduce the intraocular pressure, but in cases of exophthalmic goiter ocular hypotonus is not a feature. On the other hand, paralysis of this nerve would be expected to increase the intraocular pressure, but in clinical cases of such paralysis the tendency is in the other direction, and section of the nerve in cases of glaucoma reduces the pressure.

Whether or not the same explanation applies to glaucoma simplex as to ordinary chronic glaucoma, I am not yet able to determine. By glaucoma simplex I mean that condition, if it really exists, in which cupping of the optic disk and the characteristic visual defects of glaucoma occur with the intraocular pressure always well within the normal limits. The theory that it is due to abnormal weakness of the lamina cribrosa, and the theory recently advanced by Noiszewski that it is due to abnormally low cerebro-spinal pressure, are attractive but in my opinion are based upon insufficient evidence.

The theory that I have advanced here, is simply a tentative one. Further investigation is necessary before it can be proved or disproved. It seems, however, to explain the known facts better than any other. It is intended, of course, to apply only to the usual cases of primary glaucoma. That cases of glaucoma exceptionally occur which cannot readily be distinguished from these but which are due to some other cause or causes, I have no doubt. An important question in this connection is whether or not glaucoma can occur without any abnormal obstruction to the outflow from the eye. Judging by my histological findings I should answer this in the negative, but clinical observations suggest the contrary. A further study of the filtration angle by means of the contact glass may throw light on this question. It is a well recognized fact, of course, that peripheral anterior synechia may result from inflammatory conditions. In the absence of such synechia, obstruction to the outflow from the eye may no doubt exceptionally result from changes within the ligament itself.

ON RESULTS OF TURNING OF THE TARSUS.[1]

By Professor L. de Blaskovics, Budapest, Hungary.

(With six illustrations in the text.)

ANALYZING the causes of the generally known unsuccessfulness of operations against wrong-standing eyelashes, two main points may be emphasized in this regard. Firstly, the operations are done without caring enough for the anatomical conditions, on the other hand the cicatricial shrinking does not stop even after operation,—thus leading to recurrences.

Apart from congenital distichiasis, two abnormities in the position of the eyelashes may be chiefly taken into consideration from the operative standpoint. The first is trichiasis due to blepharo-adenitis, the other is cicatricial entropion. In the former case the shape of the tarsus is normal, whereas in the latter it is curved inwards. Therefore any kind of marginoplasty may be correct against trichiasis, as the skin-piece transplanted into the lid-margin can not touch the cornea at all. No operations of this kind may be applied, however, in cicatricial entropion, only such as strive to make the curved tarsus erect (tarsoplasty), or in which we remove the whole part of the curved tarsus (tarsectomy).

I tried a great many different methods against cicatricial entropion. Final results have been shown only in Kuhnt's tarsectomy, which I applied in many hundreds of cases. A great disadvantage of this operation lies in the resulting scar-tissue, developing in the place of the removed tarsus, the shrinking of which results in a gable-shaped extension of the eyelid from the ball. Even ugly duplicates in the whole

[1] Szemeszet (Hungarian) 1912,—*Klin. Monatsblaetter f. Augenhk.*, 1918, November.

thickness of the lid-margin may be seen after this operation. All these defects led me thirteen years ago, to recommend instead of removing the whole tarsus, the inversion of it and replacing it in this turned position.

After 300 operations with my method since that time, I had a wide opportunity to examine the exact anatomical conditions in cicatricial entropion. Before describing the technique, mode of action and results with my method, I hold it necessary to render an account of my experiences in this regard.

As is generally known, in cicatricial entropion of the upper lid, a scar-line is to be found on the conjunctiva, in the so-called subtarsal sulcus, running parallel with the lid-margin. This scar-line, so typical of cicatricial entropion is adherent strongly to the tarsus. The rest of the conjunctiva gets stretched by this shrinking process, thus being pulled towards the scar-line, the fixation-point. The tension towards the margin of the eyelid, results in the position of the eyelashes getting lower, *i. e.*, nearer and nearer to the cornea. If this tension is not the same everywhere, some eyelashes may come into a double row, or their wrong position is worse in one place, than in the other. The tarsus will grow slowly curving inwards, by the shrinking conjunctiva, lying above the typical scar-line. The form of the tarsus is not "boat-shaped"—as generally believed to be and cited in text-books—but bent in an angle along the typical scar-line.

In more progressed cases of cicatricial entropion, we find that the conjunctiva lying above the typical scar-line, is easily to be dissected away from the tarsus,—being quite impossible under normal conditions. This phenomenon was explained by Straub,[1] saying that the conjunctiva lying now above the tarsus, is the fornix pulled there gradually by the shrinking process and that what we call the typical scar-line is nothing else, than the tarsal conjunctiva shrunken to its minimum and so very strongly adhering to the tarsus.

Studying the mode of action of turning of the tarsus, in the greater number of the cases I have observed, that as soon as the typical scar-line is cut off from its base and the tarsus is freed, the curved tarsus gets straight at once. This fact

[1] *Klin. Monatsblaetter f. Augenhk.*, 1907, iv., p. 168.

proves, that the tarsus itself is not curved, only bent inwards by the pull of the cicatricial conjunctiva.

Yet we see sometimes, that the tarsus remains in its curved position, and after removing of the typical scar-line and after the tarsus is freed. Such a tarsus is very thick (many times the normal) and its vertical diameter is shorter than usual. Cases of this kind show a shrinking of the tarsus itself, but the shrinking in the horizontal direction could not develop much, as both ends of the eyelid are more strongly fixed.

It is obvious, that in milder cases no trachomatous tarsitis has preceded the cicatricial entropion, but is was present in the more severe cases. This is evidenced by Kacsó's[1] investigations, who has examined 30 tarsi removed in tarsectomy. In these histological examinations the greater number of the cases showed no traces of tarsitis, there being present at the most, infiltrations in the superficial layers of the tarsus looking to the conjunctiva.

Cicatricial entropion, in which we find only incipient shrinking, shows the best, that cicatrization of subconjunctival tissue is fully sufficient to make the tarsus curved.

Not long ago, I had a case illustrating very interestingly these conditions. The tarsus seemed to be very much thickened, the conjunctiva above it was red, rough, hypertrophic, but showing no scars as yet. The tarsus, however, had been already curved, because the eyelashes were bent inwards, a few of them even scraping the cornea. In order to free the tarsus from the overlying conjunctiva, I cauterized the latter. Scraping down the cauterized parts, I tried to execute the tarsal section, near to the lid-margin. When trying to do this, it turned out, that the tarsus supposed to be so thickened was quite normal, bearing on its surface a granulation in incipient shrinking, three times thicker than the tarsus itself. Seeing that no considerable alteration of the tarsus is necessary to be present in such colossal thickening of the subconjunctival tissue, we can easily perceive that the shrinking of the latter is quite sufficient to make the tarsus curved and fix it in this position.

In cases, however, where the tarsus was also thickened, a

[1] *Klin. Monatsblaetter f. Augenhk.*, 1922, July, p. 140.

complete entropion was generally present. No complete entropion could have been observed without this thickening of the tarsus.

These experiences led me to the conclusion, that no considerable thickening of the tarsus usually takes place in the trachomatous alterations. If the conjunctiva shrinks and this shrinking process is strong enough to curve the tarsus an entropion will result. This entropion remains incomplete. A complete entropion we shall find in cases only where a trachomatous tarsitis was present previous to the shrinking of the conjunctiva.

After this short introduction let us consider the steps of the operation of turning the tarsus. The publication is being repeated now, because modifications have been introduced since my first paper. My operation has become in its present form more complete in technique and easier in execution. The modifications refer chiefly to the fixation of the eyelid, extirpation of the scar-line and to the more effectual placing of the sutures.

At first I fixed the everted eyelid only with my finger, but found that this is not easily done, especially by beginners. Later on I modified the fixation of the everted eyelid by means of threads, recommended by Liebermann,[1] then with double-hooks and later with fork-like forceps. However, I had to give up these methods, because a partial necrosis of the lid-margin was the result of the strangulation caused by the threads. This method was not cosmetically satisfactory. Such a disagreeable complication was never observed in the following form of my operation. The modification of placing the sutures I mentioned already in the first publication, but then I recommended it only exceptionally. The below described intermarginal placing of the needle is now regularly done.

Following these suggestions my operation will be performed as follows.

Local anesthesia: Disinfection. For anesthesia I use 2*cc* of a 2% solution of novocaine containing $\frac{1}{10}$ part of 1:1000 suprarenin. We inject either under the fornix or under the

[1] *Klin. Monatsblaetter f. Augenhk.*, 1908, December.

skin, or half into both. The operation may then be begun at once.

1. *Tarsal section.* The eyelid is everted and then fixed in this position by Kuhnt's modified blepharostat, introduced from the palpebral fissure. The modification lies in an arching out of the hole-end of the instrument, thus letting free the regions of both canthi. (Fig. 1.) The cutting through the

I.

FIG. 1.

tarsus in done with a convex or hyperconvex knife, along the typical scar-line on the side close to the lid-margin, 1–1½*mm* from it. (Indicated by a thick line in Fig. 1.) The section must be made in a vertical direction through the complete thickness of the conjunctiva and tarsus. The tarsus being bent in an angle, as mentioned above—we are to be careful in directing the section vertical to the surface of the conjunctiva, lying between the typical scar-line and the lid-margin. The blade of the knife will form during the section a pointed angle with the convex-edge of the tarsus.

2. *Extirpation of the typical scar-line.* We make another section superficially, going only through the conjunctiva (represented by a thin line in Fig. 1), thus semilunarly outlining the typical scar-line. The outlined strip of conjunctiva is cut off by means of forceps and scissors. (The part to be excised is indicated by the *lined area* in Fig. 1.)

If we don't find a marked scar-line, namely in cases, where

the conjunctiva lying above the tarsus is not to be detached especially if the conjunctiva is rough, uneven, thickened and infiltrated, the whole conjunctiva, even the diseased part of the fornix is to be removed after the tarsal section. Above the arsal conjunctiva we do this by means of Liebermann's[1] cauter-excision. We cut through the conjunctiva close to the fornix, then we burn down the whole surface of tarsus with the galvano-cautery. The burnt conjunctival parts are scraped off by means of a little sharp spoon. If we are to remove also a part of the fornix, first we outline with a knife the part to be cut off and remove it with forceps and scissors, looking out, that the levator muscle lying below it, be not injured.

3. *Dissection of the conjunctiva, freeing of the tarsus and placing of sutures.* Now we are going to dissect away the conjunctiva with the convex knife. We free it from its wound-edge up to the convex edge of the tarsus. When dissecting, we should turn the edge of the knife towards the tarsus, so as not to hurt the conjunctiva. Grasping the conjunctiva with forceps is not necessary. Not much harm will result, if the tarsus be cut accidentally. In cases, where the conjunctiva is burnt down with the cautery, the dissection towards the fornix is performed with forceps and scissors. Three mattress-sutures are then placed at an equal distance from each other into the free margin of the conjunctiva. Each thread will pass two times through the conjunctiva. The threads are first passed from within the conjunctiva to its outer surface and then close to it we return the needle to the inner surface. The loops appear on the epithelial surface of the conjunctiva as shown in Fig. 2.

Thereafter we lead the knife behind the tarsus, freeing it from the pretarsal fascia by several sections. The tarsus is then grasped on its free upper edge by the fork-shaped forceps. We remove now the blepharostat, while forkshaped forceps and threads are passed through its hole-end. Then the conjunctiva is dissected away from the levator muscle by means of forceps and scissors, while the assistant is pulling up the tarsus the operator, taking the threads into his hand pulls gently down on the conjunctiva, stretches it, and thereby facili-

[1] *Arch. f. Ophtalmologie*, vol. 105, p. 542.

tates the dissection. The whole fornix must be freed, even a part of the bulbar conjunctiva up to the cornea.

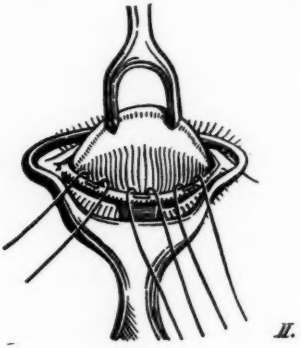

FIG. 2.

4. *Turning of the tarsus.* The tarsus is pulled down by means of the fork-shaped forceps and the assistant pulls upwards the margin of the eyelid. By this manipulation a part of the levator muscle above the convex-edge of the tarsus is exposed to view. The levator muscle is then separated on both sides of the convex tarsal edge, leaving only a pedicle, 3*mm* wide in connection with the tarsus (Fig. 3, *dotted lines*).

FIG. 3.

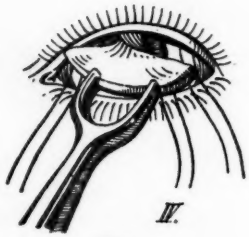

FIG. 4.

The tarsus is then revolved 180° on its pedicle (Fig. 4), so that the anterior surface will look backwards. In this turned position we replace the tarsus on its original site, meanwhile

stretching a little the threads. After replacing the tarsus, the fork-shaped forceps is cautiously removed from it.

5. *Finishing of the sutures.* Six thread-ends are now observed hanging out. Having properly arranged the threads, the needle in the middle suture is carried behind the margin of the eyelid, tilting outwards meanwhile the lid edge with our left thumb. (Fig. 5.) The needle is brought out through the intermarginal

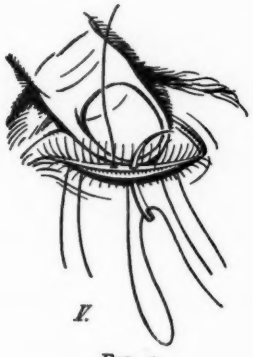

FIG. 5.

line. The other two sutures are similarly placed on either side equally distant from the middle suture. The threads are then knotted, while moderately drawing them. The margin of the eyelid is tilted outwards by this position about 90°. This position is then secured by a bandage.

The sutures are removed in 5 days. Bandaging of the eye is necessary only for 6 days. For a short time the margin of the eyelid remains turned outwards. As soon as the cicatrization of the wound has been accomplished, no turning out will be seen any more. However, I do not strive to have a permanent turning out of the lid. This is done only for securing the result. The reason of it will be discussed later on.

Externally no traces of the operation are to be seen. The position of the eyelashes will again be the normal. Eyelashes which seemed hitherto to grow out of the posterior lip of the lid-margin, have been returned to their normal position, as the tension of the conjunctival strip between lid-margin and typical scar-line has been abolished.

My operation may be executed also on the lower lid, but it is more difficult, due to the smallness of the tarsus and narrowness of the fornix. It is remarkable, that the lower fornix often gets wider after being freed from its adhesions. This may be explained by the fact that the conjunctiva is fixed to the tarsoörbital fascia in a shrunken state, the shrinking ceasing at once, after dissecting it from the adhesions.

Turning of the tarsus on the lower lid, shows only slight differences from the upper lid. No typical scar-line and no tarsus angularly bent are to be seen here. As the tarsus is very small, the tarsal section should not be farther than 1*mm* from the lid-margin. The section is to be executed very carefully. The conjunctiva is very easily to be dissected away from the lower tarsus and we are to detach it up to the cornea. Instead of the levator muscle, the fibers of tarso-orbital fascia, running to the tarsal convex edge is to be separated on both sides and the tarsus is revolved on a pedicle formed by the fascia. The suture-ends are not cut, after knotting them, but drawn down and fixed on the face with adhesive plaster. The sutures pull down the lower lid, thus resulting a temporary ectropion, which disappears as soon as the threads have been removed.

The turning of the tarsus was executed in 300 cases, four of these being on the lower lid. Serious complications did not occur during or after the operation. The only trouble lies in injuring the free end of the dissected conjunctiva during operation, or that we made a hole in it with the knife or scissors. In such cases the injured part is to be removed and the sutures are placed only thereafter.

If the conjunctiva is shortened and friable, suturing may become impossible. In such cases the freed part of the tarsus is totally to be removed. Thus we have performed tarsectomy instead of tarsoplasty. No sutures will then be necessary.

Bleeding during the operation has not caused trouble. In two cases I observed serious after-bleeding, soaking through the whole bandage. The blood-clot had to be removed and the bleeding artery had to be caught. Even ligating of the blood-vessel was necessary in the other case. However, the course of healing was uneventful in both cases.

In a few cases hypergranulation of the wound was observed.

It is advisable to wait until the hypergranulation gets more or less pedunculated, and then we cut it off with scissors or curette it with a sharp spoon. If done too early, the granulation may recur. The final result is not influenced considerably.

It occurred in one case, that a piece of the conjunctiva had slipped out into the palpebral fissure. I cut it off. The result of the operation was not affected.

I had previously referred to a necrosis of the lid-margin, occurring in those times, as we fixed the everted eyelid instead of the blepharostat, with threads, double-hooks, etc. Although no serious trouble was caused, yet the cosmetic result was not satisfactory, as no eyelashes were present in $\frac{1}{4} - \frac{1}{3}$ part of the eyelid.

A necrosis of the inverted tarsus has never occurred. However, a few times it was observed, that the tarsus slipped up after the operation, imitating a little tumor behind the skin of the eyelid. This trouble occurred in cases where the anterior surface of the levator muscle had been dissected more than necessary. In this way the connection towards the skin had been cut through. Since regarding this circumstance, no trouble of this kind recurred.

It may happen, that the wound-edges of the tarsus do not grow together and a pseudo-arthrosis-like situation arises. While everting the eyelid, the tarsus-strip is turning in- and outwards. This situation causes no trouble and may even secure a recurrence. With every contraction of the levator muscle, the fibers of which run to the pretarsal fascia, skin and tarsal strip are pulled upwards, the tarsus being tilted outwards this way.

Before enumerating statistics on results of my operation, I desire to explain the mode of action of my method. How to explain it? In the first times I thought,—and this was the starting point to apply in turning of the tarsus, that the tarsus, by the turning process of the operation, would become in the same degree curved outwards, as it was curved inward before the operation. Thus I thought the tarsus would adjust itself. When, however, I saw that the curved tarsus became straight after being freed from all adhesions, I had to give up this explanation.

The effect of the operation must lie in freeing the tarsus from

all adhesions, which kept it curved before the operation. If so, anybody could ask, is it necessary at all to turn the tarsus, straightened by freeing from its adhesions? Would it not be quite sufficient to sever the pathological adhesions? I believe, that would not suffice. By severing only the adhesions, everything would grow back in its old place, resulting in stronger scars and adhesions than before the operation, thus increasing the entropion. The results of my operation I explain by laying stress upon the fact, that after turning the tarsus, its smooth anterior surface comes opposite to the dissected conjunctiva. This will never become so adherent as previously. Even if the shrinking process is continued, it will not further influence the position of the eyelid. Upon this fact I base the definitive results of my operation.

Another postulate must be carried out, to have a successful operation. This is, that the tension of the conjunctival strip, lying between lid-margin and typical scar-line, must be abolished. This may happen from itself, as seen after tarsectomy. In order to secure the result of the operation, I found it necessary to promote in some way the retraction of the above mentioned conjunctival strip. This is done in every case by my intermarginal sutures. Fig. 6 shows, that the intermarginal

Fig. 6. *II.*

passing out of the sutures, not only pulls outwards the tarsus strip of the lid-margin, but pulls inwards the intermarginal line, drawing the abovementioned conjunctival strip in the direction indicated by the arrow.

The mode of action of my method may be explained per-

haps differently. Whether an operation is useful or not, is always to be judged from its results. Let us consider now the results of my operation.

Total number of operations (invertatio tarsi)	300 cases	
Operations made on the upper lid	296 "	
" " " " lower "	4 "	
Wrong-standing eyelashes persistent after operation	19 "	(6.3%)
Partial necrosis of the lid-margin	6 "	(2 %)
Slipping up of the tarsus	5 "	(1.6%)
Recurrence	4 "	(1.3%)

As to the 19 cases with incomplete result, I beg to remark, that the wrong position of the eyelashes was partly due to a blepharoadenitis, being of course not done away by turning of the tarsus. Such wrong-standing eyelashes have been removed by electrolytic epilation. The final result thus obtained was quite satisfactory. The failures in the other part, might have been due to faulty technique in making the tarsal section. The exact and careful execution of the operation is very important, but I emphasize that the tarsal section must be done always in the correct direction. If wrong-standing eyelashes persist in groups after the operation,—it is chiefly due to a badly directed tarsal section,—the defect will be corrected by Spencer-Watson's marginoplasty.

As to the four recurrences, one of them might have been also due to bad technique in the tarsal section. In the remaining cases the tarsus was very much thickened and in addition in one case a microphthalmus was present—a reason in itself for entropion. At any rate bad technique or a very severe state of entropion caused the four recurrences, because the failure revealed itself as early as 3–4 weeks after the operation. The remainder of the cases has shown no recurrence for many years.

Regarding the circumstance, that in three cases of recurrence a thickened tarsus was present, I decided to adhere to tarsectomy in such cases. I have, however, seldom been obliged to do this. During the period of 300 tarsus invertations,

only in eleven cases was it necessary to apply tarsectomy instead of turning of the tarsus.

I beg to mention, that even after Flarer's operation extirpation of the zone of the hair-follicles applied for entropin,—it remained necessary to turn the tarsus. Flarer's operation, so unreasonable from the operative standpoint, seems to be still performed here and there. This operation neither eliminates the trouble, nor satisfies cosmetically, and in addition mutilates the eyelid. The main trouble in wrong-standing eyelashes in entropion, does not lie in touching the cornea by the cilia. This trouble will become only disagreeable, when the eyelashes are pulled out and new grown lashes hurt the cornea. The most important evil is, however, that the skin touches the eyeball. This cannot be abolished either by Flarer's operation or by the marginoplasty. Only turning of the tarsus can do away with this condition, because the contact of skin and cornea will be eliminated by this operation.

In order to correct the cosmetic defect caused by Flarer's operation, I applied tattooing of the eyelashes. I do it in the following manner: The eyelid is anæsthetized and fixated with a blepharostat. On the margin of the lid I make numerous superficial and vertical sections, running parallel to each other with a straight keratome;—soot—produced on a piece of glass held above a burning candle (more indifferent and sterile than India-ink)—is then rubbed into the scarifications. The black lines will not fade away, and imitate very nicely the eyelashes.

My results as noted above, indicate best the advantage of turning of the tarsus over other methods. I, myself, have tried during many years a considerable number of the different methods of Snellen, Anagnostakos, Streatfield-Hotz, Panas, Müller, Kuhnt, etc.,—not speaking of the numerous kinds of marginoplasty, which I reject nowadays as operations for entropion—based upon these experiences, I decidedly must say, there is no method which can approximate the good and durable results obtained by turning of the tarsus.

MELANOSIS OF THE LIDS, CONJUNCTIVA AND SCLERA, WITH WARTLIKE GROWTHS ON THE IRIS.

By Dr. Harry Friedenwald, and Dr. Jonas S. Friedenwald, Baltimore, Md.

(With three illustrations on Text-Plate II.)

THE condition which is the subject of our report is so rare and has recently been dealt with in so thorough a fashion by Bourquin[1] that it would seem unnecessary to repeat his exhaustive review of the literature. The main interest in our case lies in its examination with the Gullstrand slit lamp and corneal microscope, and in the excellent illustrations made by Miss Susan B. Wilkes. However, we have collected references to a few cases which eluded Bourquin's review and these, together with several which have been published subsequent to his article, are noted in the bibliography below.

By way of summarizing the present knowledge of this interesting condition, which must be clearly differentiated from the "blue sclerotics" which are the result of thinning of the sclera and are associated with fragility of the bones, we may state, quoting freely from Bourquin:—melanosis of the eyes in white races is usually unilateral; sclera, conjunctiva, iris, choroid, optic papilla, lids and face may partake jointly or separately in the increased pigmentation; when the iris is involved its condition, so far as it is described by various writers, corresponds with that to be dealt with in greater detail below; melanosis of the eyes occurs most frequently in brunettes; in a few cases it has been hereditary; rarely is the visual function affected (Bourquin says never, but our case is

[1] *Zeitschr. f. Augenheilk.*, vol. 37, p. 129 and 294.

amblyopic); melanotic sarcoma seems to be the only other complication, this, too, being extremely rare.

REPORT OF THE CASE.

H. D., a white girl now aged nine, has been under our observation for four years, during which time her condition has been unchanged. The patient's father and mother and an older sister, have all perfectly normal eyes. They are all medium dark brunettes, the mother's eyes are brown, father's and sister's are blue. There is no consanguinity between the parents and no similar condition in the less immediate family so far as is known to the parents.

Except for measles and whooping cough in her second year, the child has always been quite healthy, she is very well developed physically and gets on excellently at school. A competent pediatrician reports the general examination to be completely negative except for a few carious teeth, slightly enlarged tonsils and a few palpable glands in the anterior cervical triangle. The child's hair is dark brown, her complexion rather fair. She has never fractured any bones and her blood calcium and phosphorous, examined because of the superficial similarity between her condition and the well known syndrome of blue sclerotics and fragility of the bones, were found to be normal.

Shortly after the child's birth her parents noted that her eyes "did not look right," they have observed no change in the condition up to the present except that for a short period in infancy, the child's eyes were crossed. The eyes can best be described by reference to the accompanying illustration.

The condition of both eyes is the same and very nearly symmetrical. There is an intense pigmentation of the lids which varies from a slate to a chocolate color. On the conjunctiva of the lower lids there are a few slatey patches. The scleræ show an intense bluish, almost purplish discoloration which, except for the irregular unpigmented area shown in the picture, is most intense nearest the cornea. The corneæ are quite clear, irides are dark brown and on examination with the naked eye, do not seem especially abnormal except for their uniform velvety appearance resulting from the absence of the normal trabecular structure.

With the slit lamp and corneal microscope the bulbar conjunctiva is seen to be peppered with minute irregular granules of dark brown pigment, but the main coloring is due to the great number of very fine dark pigment granules which lie in the superficial layers of the sclera and especially to the masses of pigment which lie deeper in the sclera and

ILLUSTRATING DRS. HARRY FRIEDENWALD AND JONAS S. FRIEDENWALD'S ARTICLE, "MELANOSIS OF THE LIDS, CONJUNCTIVA AND SCLERA, WITH WARTLIKE GROWTHS ON THE IRIS."

FIG. 1. The discoloration is actually many times darker than it appears in the illustration.

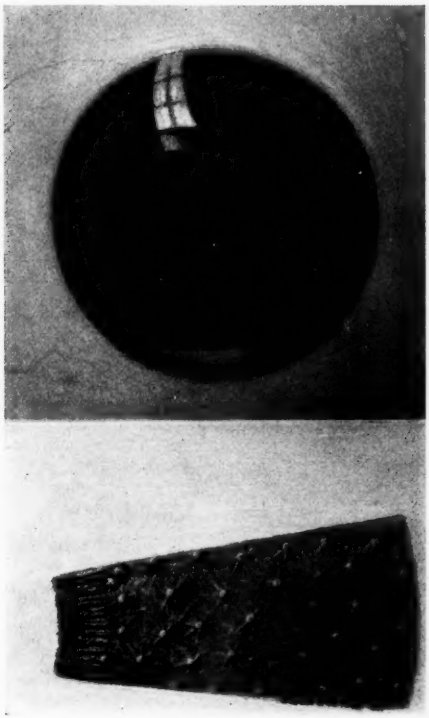

FIG. 2. Iris as seen with the corneal microscope.

cannot be clearly made out but appear bluish as seen through the overlying scleral fibers. The corneæ are quite clear, endothelial reflex observed by the method of Vogt[1] shows no abnormalities. The anterior chambers are of normal depth and quite clear. The irides are of a dark brown color, crypts and trabeculæ are absent. Perhaps the iris can best be described by likening it to a field of stubble or a deforested landscape where only the stumps of trees remain. Small cylindrical or hemispherical elevations are scattered irregularly but fairly evenly over the whole iris except for a narrow region at the pupillary margin where the iris surface is thrown into radial folds. These wart-like excrescences are of the same dark brown color as the rest of the iris. Around the base of each the iris surface shows minute folds, radial to the excrescence as if it had been puckered up in the formation of these growths.

Ophthalmoscopic examination shows the media to be clear. The sheen of light reflex from the fundi is unusually bright. The color of the fundi is that which one usually finds in brunettes, a shade which lies between orange and chocolate, but is almost impossible to describe. The optic disks are of normal color, vessels are normal. The macular region appears normal in each eye.

The whole examination is made rather difficult by an intense irregular horizontal nystagmus. Extra-ocular movements, except for the nystagmus, are normal. Pupils react actively to light and accommodation. Central vision is slightly defective—

Right Eye + 1.75 sph. + 1.00 cyl. axis 105 V = $\frac{6}{12}$.
Left Eye + 1.75 sph. + 0.75 cyl. axis 90 V = $\frac{6}{12}$.
Binocular vision = $\frac{6}{9}$.

Visual fields could not be charted on account of the unsteady fixation.

LITERATURE.

1913—Fleischer. *Klin. Monatsbl. f. Augenheilk.*, vol. li., p. 170, reports case, melanosis of one eye involving sclera, choroid and iris with wart-like excrescences on the iris.

1915—Kestenbaum. *Zeitschr. f. Augenheilk.*, vol. xxxiv., p. 317, reports case of bilateral melanosis involving face and scleræ. Irides were dark and their structure difficult to make out. (Lit.)

[1] *Atlas der Spaltlampenmikroscopie des lebenden Auges*, Berlin, 1921, p. 15.

1920—HILDESHEIMER. *Klin. Monatsbl. f. Augenheilk.*, vol. lxiv., p. 548, reports case involving conjunctiva and sclera in which the pigmentation increased while under observation.

1920—JABLONSKI. *Ibid.*, pp. 548 and 850, melanosis of sclera bilateral in two sisters.

CONSERVATIVE OPERATIVE TREATMENT OF LARGE STAPHYLOMATA OF THE CORNEA.

By Major R. E. WRIGHT, I.M.S., Superintendent, Govt. Ophthalmic Hospital, Madras.

A SUBJECT such as this will perhaps interest those ophthalmologists who work in the East, where one still sees numbers of large corneal staphylomata in the out-patient rooms, more than their Western colleagues. For some time now I have dealt with the majority of corneal staphylomata—which tended to be progressive, from slight ectasias of a few *mm* to those permanently protruding through the palpebral aperture and as much as *2cm*, in height, by trephining and cauterization. It must be understood that the method is not practiced to the exclusion of iridectomy, more especially in small ectatic scars where by this means a distinctly better optical result may be obtained and the tension is not greatly to be feared, or where there is a prospect of removing a lens at a later date. In the large majority of extensive staphylomata an iridectomy is useless. The decompression part of the operation is like the ordinary sclero-corneal trephining, except that one removes a disk at the margin of the staphyloma and puts the posterior chamber, when it exists, into direct communication with the sub-conjunctival tissues. There is, as a rule, a large escape of aqueous when the nipple-shaped protuberance collapses. The flap is replaced in the usual manner as for an Elliott's operation and then with a fine pointed electro-cautery the collapsed mass is dealt with. This portion of the operation is carried out by making a number of linear radiating eschars from center to periphery, and subsequently a more thorough umbilicated cauterization at the center. The result of the procedure is that the collapsed staphyloma shrinks up

to a reasonable size, and as there is a free escape of aqueous into the sub-conjunctival tissues during the next few weeks, there is less tendency for the newly forming scar tissue which results from the cauterization to become ectatic. Eventually the scars become more and more firm and shrink with the production of white fibrous tissue. Occasionally it happens that in extensive staphylomata a small area of transparent cornea and anterior chamber exist. Such may not be perceptible with the eye or even with the loupe, but may be readily perceived with the microscope. Should this be so, the region adjoining the clear patch is naturally selected for trephining because it may be possible under such circumstances to leave an iridectomy which gives the patient more light. In cases without even a trace of clear cornea, one cannot tell whether or not one will strike the anterior or the posterior chamber, but in the majority of these it is more than likely that the trephine button will come away with the remains of the iris adherent as a thin pigmented layer to its deep surface. One must, in doubtful cases, take the risk that the posterior chamber does exist. Should it be non-existent, and the remains of the lens and suspensory be adherent to the staphyloma, vitreous will appear at the trephine hole. If this happens we are very little worse off than before. One simply sutures the flap back in position. It is an interesting fact, however, that in the majority of those staphylomatous eyes which I have examined (and I have had the opportunity of examining a very large number in selecting our museum specimens), the posterior segment is intact or almost so, *i. e.*, the suspensory capsule partition bounds a normal vitreous in front. In many of the specimens the lens has gone, or is reduced to a thin wafer, but the suspensory and capsule still hold even though at times they are severely stretched due to ciliary distortion. It is extraordinary in how many cases one sees a perfectly normal posterior segment with healthy vitreous, retina in good position, and the suspensory capsule partition in its normal plane, whilst forward there protrudes a staphyloma almost as large as the posterior segment itself. When one remembers that this is so in a very large number of the cases, it seems almost criminal to adopt any procedure which might interfere with the integrity of the suspensory capsule barrier,

and damage the posterior segment. Most of the open operations such as ablation are qualified to do this and hence I consider that the majority of staphylomata are entitled to a trial by trephining and cauterization. Naturally the selection of the spot to trephine varies in different cases, and only in certain eyes is it possible or necessary to execute the maneuver of splitting the cornea. One merely dissects up the flap till it becomes dangerous or impracticable to dissect further. There is very little use in proceeding beyond the point at which the conjunctiva becomes firmly bound down to the rising edge of the staphyloma provided one is certain or reasonably certain of tapping the posterior chamber. The radial cauterization is an essential part of the procedure, for we thus, without exposing the interior of the globe to the danger of an unprotected wound, are able to cause a shrinkage of the tissue and set up a granulation tissue response which in time is followed by a firm scar. The after treatment of such cases is generally by instillations of saturated aqueous solution of picric acid and boric ointment pad with pressure bandage. The bandaging is kept up for several weeks.

REPORT OF A CASE OF MELANO-SARCOMA OF THE CHOROID FOLLOWED BY PHTHISIS BULBI AND IRIDO-CYCLITIS IN BOTH EYES.[1]

By Dr. Martin Cohen, New York.

(With eight colored illustrations on Text-Plates III.–IV.)

THE literature on this subject is scanty and is mainly contained in Parson's *Pathology of the Eye*, Fuchs's *Text Book*, and in the work of Leber and Krahnstover, published in 1892,[2] embodying thirty-four cases of melano-sarcoma of the choroid followed by phthisis bulbi. Schirmer,[3] in 1905, collected thirty cases of intraocular sarcoma, most of which resulted in phthisis bulbi and were reported as producing sympathetic inflammation. He claims that out of this number only thirteen were true cases of sympathetic ophthalmia. No reports of interest on this subject have since appeared and authorities differ in opinion as to whether or not melano-sarcoma of the choroid can produce sympathetic ophthalmia. Fuchs states that when irido-cyclitis follows melano-sarcoma of the choroid it is due to a necrosis of the tumor and that sympathetic ophthalmia is observed, though it is rare. Parsons, in his chapter on sympathetic ophthalmia in *Pathology of the Eye*, agrees with Leber and Krahnstover that there is not sufficient evidence to show that sympathetic ophthalmia is ever set up by sarcoma of the choroid, although in his chapter on melano-sarcoma of the choroid he states that sometimes an irido-cyclitis replaces the glaucomatous stage of intraocular growths and

[1] Read before the Section on Eye, Ear, Nose and Throat of the State Medical Society, April 22, 1924.

[2] *Archives of Ophthalmologie*, vol. xlv., 1 and 2.

[3] *Graefe-Saemisch Handbuch Der Gesamten Augenheilkunde*, vol. vi., 2 Aufl.

TEXT-PLATE III.—ILLUSTRATING DR. MARTIN COHEN'S REPORT OF A CASE OF MELANO-SARCOMA OF THE CHOROID.

FIG. 1. Section of eye in horizontal plane. The phthisical eye is compared with a normal eye, the latter outlined by the dotted circle. The wrinkled cornea is plainly seen, also the anterior synechia and much reduced anterior chamber and the post synechia. The epithelium dips into the irregularities of Bowman's membrane. Scar tissue is seen deep in the corneal stroma. The vitreous space is the small clear area behind the lens enclosed by the detached retina. The subretinal space is filled with coagulated albumin and some blood. The tumor of the choroid is seen projecting into the subretinal space and on its surface recent hemorrhages are evident. The base of the tumor is seen resting on the sclera, a small portion on the temporal side of the optic nerve and the major part on the nasal side. The faint shadows in the sclera are extensions of the tumor along the posterior ciliary vessels on the nasal side.

FIG. A. Detail showing the extensive atrophy of the ciliary processes and iris, the remnants of which are incorporated in the scar tissue extending to the lens, which is a cyclitic membrane. A portion of the anterior chamber is seen filled with blood. The occlusion of the sinus angle and Schlemm's canal is quite evident.

FIG. B. Detail showing the degeneration of the lens fibers. Morgagnian globules are seen near the posterior capsule. The delicate connective-tissue membrane lies on the posterior lens capsule which is a cyclitic membrane.

FIG. C. Detail showing tumor cells. The general spindle form and variation in pigment content is shown.

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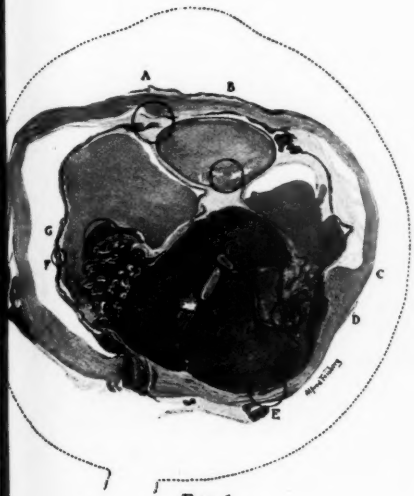
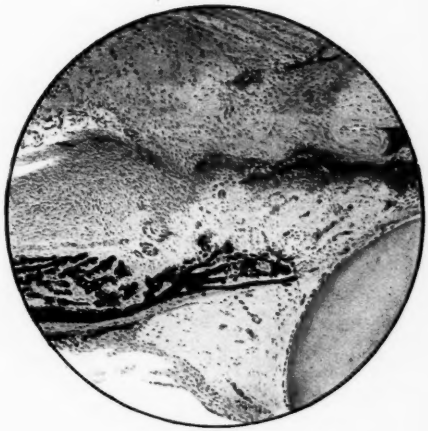


FIG. 1



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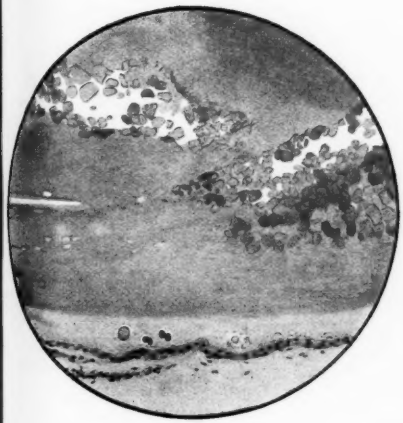

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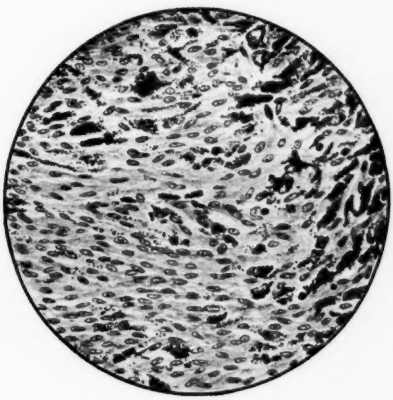

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TEXT-PLATE IV—ILLUSTRATING DR. MARTIN COHEN'S REPORT OF A CASE OF MELANO-SARCOMA OF THE CHOROID.

FIG. D. Detail showing necrosis of tumor mass. The narrow slits indicate cholesterin crystals.

FIG. E. Detail showing the extension of the tumor about the posterior ciliary vessels on the nasal side of the optic nerve.

FIG. F. Detail showing the distended vessels of the choroid and the numerous polynuclear leucocytes within them, and many of the leucocytes are eosinophiles. The pigment epithelium layer shows proliferation and exfoliation into the subretinal exudate.

FIG. G. Detail showing the surface of the tumor. The distended vessels of the choroid at the surface of the tumor have evidently ruptured and the hemorrhage has escaped into the subretinal exudate through the ruptured glass membrane. The choroid stroma between the vessels shows beginning necrosis.

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D

E

F

G

manifests all the signs of the simple inflammatory disease and, like it, goes on to shrinkage of the globe; there is, he states, evidence to show that it may cause sympathetic ophthalmia. Schirmer is of the opinion that intraocular tumors do not cause sympathetic ophthalmia but believes that the binocular iridocyclitis following intraocular tumors is due to an independent systemic infection.

These contrary expressions of opinion on this subject indicate that further reports in the literature are essential in order to establish definitely whether intraocular sarcomas can be the primary cause of a sympathetic ophthalmia.

CASE REPORT.—J. T., male, 35 years of age; nativity, United States; occupation, accountant; was admitted to the Hospital for Joint Diseases on February 10, 1922, on the service of Dr. Frauenthal, with a diagnosis of multiple arthritis. He complained of pain and puffiness of both ankles, also of general pains, and there was a moderate urethral discharge. His family history was negative; his past history showed that he had always been in good health until 1916, when he had an acute attack of appendicitis for which he was operated upon with an excellent result.

Venereal History.—Seven years ago he had an attack of gonorrhoea, for which he was not treated by a physician. Three years later he had another attack, and a few years later a third attack. For the last two attacks he received medical care. He had a chancre ten years ago, for which he received mixed treatment for two years. During this period, according to the patient's statements, the blood and spinal Wassermann examinations and tuberculin tests were negative. Early in the course of his syphilitic infection he received half a dozen salvarsan injections.

Present Illness.—On admission to hospital the blood Wassermann and gonococcal fixation tests were negative. The patient was bed-ridden on account of his arthritis, looked pale and thin. He slept well, however, his bowels were regular, his appetite was excellent, his pulse and respiration were normal. He had a slight urethral discharge; his right ankle had been œdematous and painful for three years and his left ankle had been in a similar condition for the past year, during which time he had suffered pain in all his joints as well as pain in the heels when walking. Examination of the prostate indicated a nodular mass in both lobes; the vesicles were palpable; the prostatic secretions showed pus cells but no gonococci. The physical examination was normal except for the conditions already mentioned.

The blood report showed: Hæmoglobin, 75 per cent.; red blood cells, 4,600,000; white cells, 8,000; polymorphonuclears, 71 per cent.; small lymphocytes, 23 per cent.; large lymphocytes, 5 per cent.; transitionals, 1 per cent. The urine was negative.

After the patient had been in the hospital for three days he complained of poor vision in his left eye, and I was called in to make an ophthalmological examination, which was as follows: The right eye was normal in every respect. In the left eye the vision was possible only in the lower field, where it was limited to counting fingers at more than twenty feet. The field of vision was deficient above, nasally and temporally. Intraocular tension taken with Schiotz's tonometer was 20*mm* of *Hg.* or normal. Transillumination showed a slight shadow in the pupil from below. The media were all clear. The anterior chamber was normal. The fundus examination revealed a sharply defined large ovoid grayish mass, more than double the size of the papilla, which protruded seven diopters, at its high point, into the vitreous. This mass started about two and a half disk diameters below the papilla; a slight detachment of the retina was noted at its edge, and the retinal blood vessels made an abrupt angle at the margin and coursed smoothly over it without evidence of any regular indentations in their course. Pigmentary spots were seen between the blood vessels coursing over this mass, and no motility over the growth could be observed. The fundus was otherwise normal,—which included examination of the papilla, macula, and vasculature.

The orthopedic diagnosis was multiple arthritis, probably of gonococcal origin; other foci of infection, such as the sinuses, tonsils, teeth, and so forth, were not considered as etiological factors. Treatment consisted of hot packs, blue ointment to ankles, and internal treatment.

The ophthalmological diagnosis was melano-sarcoma of the choroid. Immediate enucleation was advised but was refused. Several subsequent eye examinations were made, one of which, on March 10th, showed the left upper lid to be slightly œdematous, with conjunctival congestion. This inflammatory reaction lasted for a few days. The fundus examination was the same as previously, except for a slight peri-papillar œdema. The patient was discharged from the hospital on March 21, 1922, with slight improvement in his joint condition. After leaving the hospital he went to his home where most of the time he remained in bed on account of his joint involvement. I did not see him again for ten months, when he sought admission to the Manhattan Eye, Ear and Throat Hospital because of the pain and the deterioration of vision in the sarcomatous eye.

He was admitted to the hospital on January 26, 1923, on Dr. Wootton's service. His physical condition had not altered since his last examination, but he stated that six days previously he had had acute pain and inflammation in his left or sarcomatous eye and that three days later he had been unable to see from it. On admission, the right eye was normal in every respect. The left eye showed œdema of the lids; the conjunctiva was slightly chemotic; the cornea had a steamy appearance; the iris markings were indistinct; the pupil was moderately dilated; the focal illumination showed a grayish reflex from the pupillary area; the intraocular tension taken digitally was apparently increased, and there was some extraocular pain.

On February 2, 1923, the œdema of the lids and chemosis subsided, but the cornea was still cloudy, the iris dilated irregularly under atropine, the intraocular tension was still apparently increased, while with focal examination the fundus reflex could be established although no details were evident and there was still some extraocular pain. Four days later blood appeared in the anterior chamber. On February 12, 1923, the cornea appeared to be normal but the anterior chamber still contained blood. Otherwise the external examination,—such as the iris, pupil, and the intraocular tension taken with the tonometer,—was normal. The fundus examination showed the same characteristic growth protruding into the vitreous as was originally seen in the eye, without any apparent change in its contour or in the surrounding area. The inflammatory changes present, mainly in the anterior segment of the eyeball, were probably due to a temporary mild iridocyclitis and glaucoma, secondary to a necrosis of the tumor mass and also to several hemorrhages. The right eye was still normal.

He was discharged from the hospital on February 26, 1923, with improvement in his eye condition. Enucleation was not performed because of a difference of opinion as to the cause of the intraocular mass. It was suggested that because of the long standing of the condition without any evidence of marked glaucoma and because of the complicated general and focal infections the case might possibly be of inflammatory origin and not of a sarcomatous nature.

He then went to Long Island to recuperate, where he remained for most of the time in an invalid chair because of the joint condition. After three weeks, he again had an attack of acute pain in his left or sarcomatous eye, which was associated with ocular inflammation and was not due to any trauma. The pain and inflammation recurred several times, and then the eye gradually shrank. Three months later the patient complained of deterioration of vision in

his right or previously normal eye and he was then readmitted to the Manhattan Eye, Ear and Throat Hospital on July 17, 1923, under the care of Dr. Van Fleet.

The diagnosis on admission was phthisis bulbi, left eye, and plastic irido-cyclitis, right eye,—the general physical condition remaining the same as previously reported. The eye examination on admission was as follows: The left or sarcomatous eye had no light perception, was soft, markedly shrunken, quadrilateral in shape, and devoid of any pain; the ocular conjunctiva was slightly injected, the motility of the eyeball was impaired, the cornea was small and cloudy. The right or second eye had a vision of $\frac{15}{20}$, with slight ciliary injection; punctate deposits were present on Descemet's membrane; the anterior chamber was normal. There were pigment deposits on the anterior capsule of the lens and diffuse opacities in the vitreous. The pupil dilated irregularly with atropine, due to posterior synechia, the intraocular tension was apparently normal digitally and the fundus details were not discernible, due to the vitreous opacities. Enucleation was now advised and accepted for the phthisical eye, on the ground that the condition might be of sympathetic nature and that its removal would have a beneficial effect on the other or plastic iridocyclitic eye.

The phthisical eye was enucleated on July 17, 1923, and was placed in a five per cent. formalin solution for future microscopical study. The second or remaining eye gradually grew worse from every standpoint, and after two weeks in the hospital became reduced to $\frac{1}{200}$; the ciliary injection, the deposits on Descemet's membrane, and the pigment deposits on the anterior lens capsule were increased in quantity; the dust-like opacities became more flocculent and dense, while the intraocular tension remained normal.

The patient was now referred to the radiologist who reported that there was an area of absorption around the roots of certain teeth,—which were afterwards removed; that the sinuses were all extremely large, and that with the exception of a slight thickening of the membrane in the frontal, the sinuses were clear. The laboratory report of the blood Wassermann and gonococcal fixation tests were both negative, and the urine examination was normal.

The patient received instillations of atropine and hot compresses to the remaining eye, as well as energetic aspirin treatment, associated intermittently with sterile milk injections and gonococcal vaccine for several weeks, without altering the progress of the plastic iridocyclitis. He was then transferred to the nose and throat department of the hospital under the care of Dr. Wilson, as it was considered that a sinusitis might be an etiological factor in the causa-

tion of the irido-cyclitis and that it might also be the cause of the multiple arthritis. The ethmoids and antra were opened and curetted intranasally, and both contained polypi and pus,—more especially the right antrum,—and a microscopical examination of the bone fragments showed an osteitis. The sinuses were treated locally for about a week, without any improvement in the eye condition. The patient was naturally discouraged with the gloomy outlook, and asked to be discharged. He went home on the 5th of August, 1923, where he was visited several times by Dr. Houghton, the attending internist of the hospital. Dr. Houghton's report of one month ago,—which is six months after his discharge,—as to his physical examination was that it showed a normal condition of the heart and lungs, no apparent palpable enlargement of liver and spleen, nor any external manifestation of metastasis, and that the multiple arthritis and the vision were not improved.

According to a report obtained from the mother ten days ago, the vision of her son's eye had rapidly deteriorated since he left the hospital, and was then reduced to the recognition of motion fingers at two feet. I have been unable to make an ophthalmological examination of the patient at home, as his family is discouraged and object to having him submitted to any further examinations.

Although sympathetic ophthalmia secondary to melano-sarcoma is extremely rare in the absence of systemic and focal infections, it remains a fact that such cases have been reported. It is known that systemic and focal infections can produce binocular plastic iridocyclitis, and its occurrence in this report naturally complicates the case; still, I believe that even if these infections could be proved to exist they would not have produced a picture such as is presented in this case.

After the proper fixation of the eye in formalin, it was embedded in celloidin, then sectioned, and some fifty sections from various levels were made, using eosin-hæmatoxylin, Van Gieson, Gram, and carbol-fuchsin stains for microscopical study. An examination was also made for micro-organisms,—such as tubercle bacilli and other organisms,—in the tumor mass and eye structures, but none were found. The examination of the sections showed no evidence of any perforation of the eyeball, which fact would probably exclude any exogenous infection. The microscopical eye findings were as follows:

Cornea.—The anterior cells of the epithelial layer took the hæmatoxylin stain badly, owing to slight degeneration. Bowman's membrane is folded and absent in places, allowing the basal cells to pass into the lamellar structure, and

epithelial cells are seen beneath it. The substantia propria has lost its regular lamellations and a band-like scar tissue is seen below Bowman's membrane near the limbus. Blood vessels appear in this scar tissue and stroma. Polynuclear cells are seen in the vessels and round cells about them. Descemet's membrane is considerably wrinkled and convoluted and its endothelium is absent in places.

Sclera (anterior portion).—The anterior ciliary vessels are filled with polynuclear leucocytes and eosinophiles; the stroma is filled with round cells, plasma cells, and polynuclears, especially eosinophiles. These cells appear to pass through to the conjunctival epithelium, and this inflammatory reaction extends to the muscular insertions.

Schlemm's Canal.—This is closed to a great extent but in some places it is filled with polynuclears and red blood cells. Fontana's spaces are compressed and infiltrated with pigment bearing cells, and the pectinate ligament is atrophic.

Anterior Chamber.—This is practically obliterated, due to an almost complete anterior synechia, and the filtration angle is filled with blood.

Iris.—Its anterior layer is adherent almost entirely to Descemet's membrane and an anterior inflammatory pupillary membrane, consisting possibly of fibrin, is seen. The stroma is largely replaced by scar tissue for about one half of its surface, and the remaining portion is atrophic and infiltrated with round cells, plasma cells, polynuclears, eosinophiles and pigment bearing cells which show marked migratory reaction.

Posterior Chamber.—This is almost obliterated except for a small space which is filled with scar tissue. This scar tissue extends entirely about the lens and is very dense on the side, where it largely replaces the iris and ciliary processes. This membrane is thin behind the lens and reaches the ciliary processes of the other side.

Ciliary Body.—One side shows practically complete cicatrization of its structure affecting nearly all of the muscle fibers on one side, while on the other side the various muscle fibers are preserved. The scar tissue extends to the pars planus ciliaris, involving the hyaloid membrane and zonule fibers, and atrophic areas are seen in the body and in the processes.

Lens.—This is swollen, surrounded by a cyclitic membrane: its fibers are broken and the spaces are filled with granular debris and Morgagnian globules, producing a complicated cataract.

Vitreous.—The cavity shows a marked reduction in its size, due to the atrophy of the eyeball and the encroachment of the intraocular tumor. It is surrounded by the cyclitic

membrane and the detached retina, and in the cavity are to be seen a few polynuclears, endothelial cells, and pigment bearing cells.

Retina.—It is completely detached and folded upon itself and is attached anteriorly to the cyclitic membrane and posteriorly to the tumor, into which at intervals it indents. It is markedly infiltrated with blood, pigment, and scar tissue, and areas of necrosis are seen. The pigment epithelium shows proliferation and exfoliation, especially over the drusen of the lamina vitrea. The sub-retinal space is filled with coagulated albumin, pigment cells, and a recent hemorrhage.

Choroid.—This is almost entirely replaced by the tumor mass, and where the tissue is seen it is engorged, the vessels being filled with numerous polynuclears, most of which are eosinophiles. Anteriorly and nasally, the choroid and ciliary body are detached, allowing albumin and blood to be seen in the suprachoroidea.

Tumor.—It practically embraces about two-thirds of the interior of the eye; its base is on the sclera, its surface is nodular, and it is made up of closely arranged spindle cells, most of which are pigmented, while it also contains small and large blood channels with extravasations. Cellular debris, indicating necrosis, is mainly seen in the central portion of the growth where the crowded neoplastic cells have been cut off from their blood supply. Cholesterin crystal formations are to be seen, signifying that the pathological process is of long standing. Bruch's membrane can be traced over the summit of the tumor, where a rupture exists allowing a recent hemorrhage to extend into the subretinal exudate. At the margin of the tumor the neoplastic cells are seen invading the stroma of the choroid. *Sclera.*—Thickened and tumor cells extend around posterior ciliary vessels.

Optic Nerve.—Atrophy is indicated, with increase of the neuroglia and connective tissue septa. The dural sheath is thickened and the subdural and arachnoid spaces are obliterated, due to endothelial proliferation.

A summary of the microscopical eye findings is as follows:

1. Melano-sarcoma or chromatophoroma of the choroid with necrosis. 2. Keratitis degenerativa. 3. Iridocyclitis plastica. 4. Complicated cataract. 5. Secondary glaucoma. 6. Detached retina. 7. Recurrent hemorrhages. 8. Congestion and detachment of choroid and ciliary body of one side. 9. Sub-retinal exudate. 10. Chronic inflammation of the optic nerve with atrophy.

There is presented an acute exacerbation of the chronic inflammation, but the pathological picture is not that of a fully developed sympathetic ophthalmia, for the characteristic numerous lymphocytes and the epithelial cells and giant cells are absent.

A summary of the facts of the case report is as follows:

1. The patient presented a multiple arthritis, with an old history of lues and gonorrhea which gave systematic negative laboratory reports.

2. There was present a melano-sarcoma of the left eye which became necrotic in areas and which probably produced a plastic iridocyclitis and phthisis bulbi, and after three months these conditions were followed by a plastic iridocyclitis in the other eye, suggesting clinically a sympathetic ophthalmia.

3. Microscopical specimens showed no perforation of the eyeball, probably excluding an exogenous infection, and no micro-organisms could be demonstrated in any portion of the eye, probably ruling out any microbic infection. The sarcomatous eye showed no histological findings of sympathetic ophthalmia.

4. It is unlikely that the systemic or focal infections caused the plastic iridocyclitis in either eye.

(a) Because of the long period during which the laboratory findings for the infections remained uniformly negative.

(b) The long delay of the occurrence of any acute eye manifestations after a history of initial infection.

(c) Because of the length of the interval, namely, three months, between the involvement of the first and second eye.

5. If, however, the multiple arthritis is of unknown origin, it is possible that this unknown infection could produce a plastic iridocyclitis in the sarcomatous eye, which is less resistant than the other eye, and that after months this other eye could become involved in a plastic iridocyclitis from this unknown infection.

6. But it is, I believe, probable that the necrosis of the melano-sarcoma was the primary cause of the plastic iridocyclitis and phthisis bulbi, which in their turn probably acted

as the exciting cause for the plastic iridocyclitis in the other eye, suggesting clinically a sympathetic ophthalmia. This conclusion is based upon the following reasons:

(a) The presence of a plastic iridocyclitis in both eyes.
(b) The sequence in the clinical pathology of both eyes.
(c) The absence of any definite proof of any systemic infection which, if present, would, as a rule, have produced a plastic iridocyclitis in both eyes nearly simultaneously.

7. The fact that melano-sarcoma of the choroid may produce a sympathetic ophthalmia, would be an additional reason for advising early enucleation, as metastasis is apt to be delayed for years, while sympathetic ophthalmia is usually a question of only weeks or months.

I wish to express my sincere thanks to Drs. Wootton, Van Fleet, and Houghton for their kind coöperation, and to Dr. Meeker for her able assistance in the microscopical report of these cases.

INFLAMMATORY SWELLINGS SIMULATING DACRYOCYSTITIS.[1]

By Dr. John Green, Jr., St. Louis, Mo.

AN inflammatory swelling in the skin over the site or in the immediate neighborhood of the lacrimal sac is, not rarely, an incident in the course of purulent dacryocystitis. A long continued low grade inflammation dependent on deficiency in drainage may suddenly assume an acute character and take on the well-known picture of a lacrimal abscess or phlegmon.

These perfectly obvious cases should not blind us to the possibility that such swellings may have other origins. The close proximity to this region of the frontal and ethmoidal cells and the extreme frequency with which these sinuses are involved in purulent inflammations would suggest *a priori* that dehiscences in the thin bony walls which separate these cells from the anterior part of the orbit might permit the extension of such inflammations to the region of the lacrimal sac. Well authenticated cases of this character are, no doubt, within the experience of every member of this Society.

In this connection anatomical studies on the relation of the lacrimal fossa to the ethmoidal cells are of great interest. Whitnall (1) found that the so-called "lacrimal ethmoidal cells" which come into relation with the lacrimal fossa belong to the group of anterior ethmoidal cells; their walls are completed by the lacrimal and frontal bones, and, in addition, often by the frontal process of the maxilla. When well developed they may enclose the lacrimal fossa on its anterior and medial as well as posterior aspects: they open into the ethmoidal in-

[1] Read before The American Ophthalmological Society, June 17, 1924.

fundibulum. The anterior ethmoidal cells, while primarily confined to the lateral ethmoid mass, very often extend into the body of the middle turbinate bone, the superior part of the uncinate process of the ethmoid bone and the agger nasi (the ridge at the superior extremity of the uncinate process of the maxilla with which the anterior extremity of the middle turbinate bone articulates). A cell in this last situation, at the superior extremity of the uncinate process of the ethmoid bone (the cell of the agger nasi) has been found very frequently related to the lacrimal fossa.

Of one hundred skulls examined by Whitnall approximately the inferior half of the lacrimal fossa was found in every case related solely to the anterior part of the middle nasal fossa, which could easily be entered through the thin posterior portion of the area formed by the lacrimal bone, while the superior half of the lacrimal fossa presented relations to an anterior ethmoid cell which extended (1) to the posterior wall of the fossa in 14 cases, (2) as far forward as the center of the medial wall in 32 instances, (3) completely across behind the fossa in 54 cases. Thus the posterior lacrimal portion of the upper half of the fossa was related to an ethmoidal cell in every case. There was, in most cases, a single ethmoidal cell situated in the anterior extremity of the uncinate process of the ethmoid bone, in the region of the agger nasi. This cell opened into the ethmoidal infundibulum.

Under the title "Peridacryocystitis" Elschnig (2) has described a group of cases, hardly to be distinguished clinically from phlegmonous dacryocystitis, in which inflammation extends from the mucous membrane of the nose or sinus by continuity or through a communicating vessel to the neighborhood of the tear sac. In such cases the lacrimal canal is permeable, and in the early stages the whole process may be cured by freely opening the ethmoidal cells or by appropriate nasal treatment. If unchecked, the anterior wall of the sac becomes eroded and we have the clinical picture of lacrimal abscess.

Rhese (3) states that diseased anterior ethmoid cells have much the same pathological significance for the lacrimal sac as diseased posterior sinuses have for the orbital contents. The prognosis of dacryocystitis of ethmoidal origin is excellent, the ethmoid operation proving curative even in very obstinate

cases with fistula. He insists that in every case of dacryocystitis radiographs should be made of the ethmoidal cells (with a probe in the nasal duct) and that, if there is evidence of disease, these cells should be thoroughly curetted.

Another variation in the clinical picture is presented by the class of cases described by Peters (cited by Posey) (4) in which there are all the signs of abscess, and the sac itself appears dilated, but where the Bowman incision and the passage of probes into the nose does not release pus, the purulent matter escaping only after the probe is withdrawn and passed horizontally into the cells forming the inner orbital wall. In these cases there is actually an ectasia of the sac, which is occasioned by stoppage of the orifices of the sac by the pressure exerted externally by pus which comes either from an empyæma of the frontal or ethmoidal cells, from the maxillary antrum or from a maxillary periostitis excited by a carious tooth.

In this connection one possible source of diagnostic error should be pointed out. It is generally assumed that permeability of the lacrimal canal to fluids is good evidence of the absence of disease of the sac and duct. V. Szily (5), under the title "Fistula Interna Sacci Lacrymalis" has described a false passage arising spontaneously, extending from the lacrimal sac to the nasal cavity, the nasal orifice occurring at the level of the middle turbinate. Thus fluid injected through the punctum appears at the nostril, though there may coexist complete stenosis of the duct. Such a fistula is ascribed to erosion of the medial wall of the lacrimal sac by empyæma of the ethmoidal cells or to perforation due to tuberculosis of the sac.

Purulent collections in the deeper parts of the orbit may "by the peculiarity of the insertion of the fascia" gravitate to the region of the sac (Posey) (4), thus simulating a lacrimal abscess. The true nature of such cases could hardly mislead an ophthalmic diagnostician.

A furuncle may occur over the site of the lacrimal sac, as elsewhere in the skin. Villard (6) finds scant mention of this condition in the literature. He has seen twenty cases among 32,000 patients, but believes that the incidence is much higher as many such patients never come under the observation of an oculist. It is always unilateral and usually not accompanied

by furuncles elsewhere. The differential diagnosis offers no difficulties.

Fava (7) reports a case of ulcer over the site of the lacrimal sac associated with abscesses of the cheek and lower jaw. The patient was a peasant, free from syphilis and tuberculosis. A probe could be passed from the base of the ulcer into the lacrimal sac whence issued thick cheesy pus. The left preauricular and cervical glands were enlarged. Cultures yielded colonies of a filamentous fungus belonging to the species Nocardia. The lesion healed after the use of iodine, locally and internally.

That, rarely, a gummatous inflammation at the inner canthus may simulate a dacryocystitis is suggested by a case reported by Luedde (8). His patient was a female, age 36, who presented a swelling over the lacrimal sac diagnosticated (in an Eye Clinic) as dacryocystitis and incised. As the condition did not improve, the patient consulted Luedde who found the tear passage freely permeable. Nodular swellings soon appeared near the lower punctum. The condition finally yielded to large doses of potassium iodide.

Case 1.—M. A., a healthy female infant of four months was brought to me November 9, 1923. Apart from a tedious labor (the mother is a primipara) there was nothing worthy of note about the child's birth. Always healthy, and had been gaining in weight. Breast fed.

Four days prior to the consultation, the mother noticed a slight swelling just below the internal canthus of the left eye. As she had a coryza, she assumed that she had given the child a "cold in the eye." The swelling increased in size, and the child became restless and feverish.

Examination revealed a rounded swelling just below the level of the internal canthus. The overlying skin was red. Fluctuation was present.

The following day an attempt was made to enter the left lower punctum, the attempt failing because of the tenseness of the lid-margin. A deep incision was made, releasing about half a teaspoonful of thick, creamy pus. A moist bichlorid pack was placed over the incision. The following day the abscess was draining freely, and the moist dressing was re-applied. Subsequently a small rubber drain was inserted daily. The routine daily treatment consisted of irrigations of the cavity with normal saline solution followed by mercurochrome 220, two percent., and drainage.

Under this treatment the pus rapidly diminished and on November 25th it was thought proper to omit the drain. At this time an exploration of the cavity with a probe revealed the presence of roughened bone. The cavity extended downward almost to the alveolar process, nasally about 5*mm*, and temporally about the same distance. The probe met with resistance when directed upward and inward toward the region of the sac. A No. 2 Theobald probe was passed to the nostril through the lower punctum. The probe slid through the bony duct without resistance. There was no evidence of denuded bone. With the probe *in situ* another probe was entered in the abscess cavity. Exploration with the second probe failed to elicit the metallic click that I expected; there was no evidence that the two probes came in contact.

Subsequently, two per cent. methylene blue solution was injected through the lower punctum. The solution immediately appeared in the nostril but did not come out through the opening in the skin. The baby had the benefit of a careful general examination by Dr. T. C. Hempelmann, with negative results. Nasal examination (Dr. Pfingsten) showed a bulging anterior ethmoid cell on the left side but no pus. It was Dr. Pfingsten's opinion that conditions in the nose failed to explain the abscess. X-ray picture of the head was negative. Wassermann negative. The child was given mercury with chalk gr. $\frac{1}{6}$ *t.i.d.*, and Dakin's solution was used for irrigation. This treatment was begun December 22, 1923, and was followed by very rapid improvement, so that shortly after January 1, 1924, the opening closed permanently.

COMMENT.

In the absence of a rhinogenic and dental origin there is no escaping the conclusion that a localized focus of osteomyelitis of the superior maxilla gave rise to a subperiosteal abscess which happened to form at the usual site of a lacrimal abscess.

I am indebted to Dr. T. C. Hempelmann, a pediatrician, for permission to report the following case.

CASE 2.—J. S., a female, aged eight months, a well-nourished and well-developed child, was first seen July 7, 1923, with a swelling situated below the left eye and near the nose. The area, which was slightly reddened, resembled the swelling of a lacrimal abscess. No abnormality could

be made out in the nose or throat. T. 99 F. On July 14th, the appearances were similar except that the swelling was larger and harder. T. 99.4 F. The mouth, gums, throat and nose appeared to be normal. Suspecting that there might be an osteomyelitis of the superior maxilla, the child was sent to a rhinologist, Dr. C. A. Gundelach, for nasal examination, which proved entirely negative. The child was also seen by Dr. Wm. F. Hardy who was quite satisfied that there was no inflammation of the tear sac. The patient was then referred to a surgeon, Dr. M. B. Clopton, who made a small, superficial incision in the cheek over the site of greatest tumefaction, but failed to find pus. The cheek became more swollen and a slight œdema of the eyelids was shortly afterwards observed. Two days later Dr. Clopton plunged a knife down to the bone and yellow pus welled from the wound.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. Dickinson, London.

The opening meeting of the Section was held on Friday, October 11th, under the presidency of Sir Arnold Lawson, K.B.E.

Case of Hole (?) in the Optic Disc.

Mr. Rayner Batten showed a patient with a peculiar appearance in the optic disk, which might be due to either a swelling or a depression. It was situated behind the scleral ring. Possibly it might be a clear-walled cyst.

The President's view was that it was a hole, with a vessel running across the bottom of it. There was a strong parallax.

Mr. Treacher Collins thought it might be a crater-like depression filled with neuroglial tissue, such as described by Coats.

Mr. Juler agreed with the President's view, and referred to the findings of Mr. Coats in his cases.

The President's Address: Tints and their Values.

Sir Arnold Lawson then delivered his Presidential Address. He said the subject of tints had become a somewhat prominent one recently, and much had been written—some reasonably, some unreasonably—about the destructive effects of ultra-violet rays on human eyes. Owing largely to alarums for which the optical trade was responsible, the public seemed to have become largely imbued with a haunting fear of day-

light, so that a large body of healthy people were wearing tinted glasses for protection. These glasses seemed to Sir Arnold to have been prescribed in an indiscriminate and indefinite way.

Assuming the wave-theory of light to be correct, it was known that the visible spectrum extended from waves of light of 700μμ at the red end to 400μμ at the violet end. The infra-red rays could pass into the eye unimpeded, and the ultra-violet rays caused harmful chemical action. The eye possessed no direct protection from the luminous and infra-red rays, but Nature provided a good deal of protection against the ultra-violet rays. Experiment showed that this protection was furnished by the cornea and the crystalline lens, especially the lens; the cornea absorbed all waves of shorter length than about 290μμ while the lens was impervious to ultra-violet rays of shorter wave-length than 350μμ. In the laboratory ultra-violet radiation had been traced to waves of about 20μμ hence only a very small proportion of possible ultra-violet radiation could possibly reach the retina normally. And ordinary sunlight at low altitudes was rather poor in ultra-violet rays; it contained few of shorter wave-length than 350μμ. Also, protection from the heat radiation at the red end was to some extent afforded by the absorptive properties of the pigment lining the back of the iris and choroid, and by the pupil's power of contraction. In all human races the eye was self-adapted to endure without harm the sunlight of the particular part of the world they lived in. Hence healthy eyes, in ordinary circumstances, did not need protection by tints.

The situation was, however, somewhat complicated by various subsidiary factors, the most important being (1) the intensity of the light, (2) the duration of the exposure, (3) the presence of glare, (4) occupation or trade.

With regard to the first, extreme degrees of sunlight, especially in the rarified atmosphere of mountains, are abnormally charged with ultra-violet and infra-red radiations, and this implies an increased dosage. The chief danger from the eye experiencing intense sunlight seemed to be from the red and not from the violet end of the spectrum.

Concerning the duration of the exposure, under certain

conditions of health, prolonged exposure to even moderate degrees of light may be harmful, due to a lowering of the natural resistance.

As to glare, this may considerably complicate the patient problem. Glare was the unpleasant effect caused by excessive reflection from below. Usually glare proceeded from snow, desert sand, rock, water, or chalk. The light, in glare, struck the eye in an unaccustomed manner, and it was something beyond normal experience. Snow reflected about 70% of the existing sunlight, and snow enabled the effects of intense light even when the direct sunlight was of but moderate intensity. The effects of glare were exerted more in the luminous and infra-red regions of the spectrum than in the ultra-violet; hence it was reasonable to consider snow-blindness as due not so much to excess of ultra-violet radiation as to excess of exposure to the luminous and infra-red radiations. The conjunctivitis accompanying snow-blindness was probably due to sunburn of the conjunctiva.

Dealing with the effects of occupation, he said that such trades as acetylene-welding exposed the worker to intense ultra-violet radiation; and, on the other hand, glass-blowers and workers in foundries were subjected to excessive heat-radiation.

A Consideration of Tints.

Sir Arnold then passed to a consideration of the spectroscopic values of various tinted glasses. For this study, he selected London smoke No. 3, three shades of amber, five shades of Fieuzal, Crookes' A, A2, B, B2, and Peacock Blue. All the glasses so tested were of *2mm* thickness, except that Peacock Blue was examined in thicknesses of 5 and *2mm.* Thickness of glass was a most important point.

The experiments were arranged by Mr. Hamblin, and the spectroscopic photographs he was showing were executed by Dr. Judd Lewis, Professor of Physics at University College. These latter were embodied in a chart shown.

With regard to ultra-violet radiation, the greatest absorptive power was noted from the darker Fieuzal tints, while the Peacock Blue and the Crookes' glass produced much the same effect. Next in order came amber shades, and the

least protective was No. 3 shade of London smoke. All the Crookes shades had practically the same absorptive power, and none of the Crookes' shades protected the eye against wave-lengths shorter than 350. The only tints which cut off all ultra-violet radiation were the darker shades of Fieuzal. For acetylene-welding the darker shades of Fieuzal were much better than Crookes' glass, and in high altitudes and for snow-blindness nothing gave so much protection and comfort as Fieuzal 4 and 5 or Peacock Blue.

Concerning red and infra-red radiation, the Peacock Blue was by far the most effective, especially when *2mm* thick, next in order being Crookes' B2 and Fieuzal 4 and 5. The various shades of amber were less effective than the corresponding shades of Fieuzal, and less so than a medium shade of London smoke or Crookes' B2.

All the various kinds of tint were equally effective in generally diminishing the luminous spectrum. Prescribers of tints should always pay special attention to the thickness of the tinted glass. Another series of photographs were taken showing the absorption of the tints when of only .05*mm* thickness. The absorption effect, in the case of the thin glass, was greatly lessened. A glass of less than *2mm* thickness was likely to produce an effect considerably less than was expected. This consideration was of special value when ordering a tint without any incorporated lens, and when ordering a tint to be overlaid on a minus lens. In these circumstances it was well to specify the thickness of glass to be employed. It would be well if some scientific body, such as this Section, could agree on uniformity of glass for prescriptions for these tints. No such uniformity existed at the present time.

He then passed to a consideration of the adaptation of these principles to the wearing of tints in health and disease.

Tints for Healthy Eyes.

Under ordinary circumtances, protection by ultra-violet glasses was quite unnecessary. When extraordinary circumstances were apt to be encountered, the two main factors to be considered were the luminous and the heat radiations, rather than the ultra-violet. In high sunlight and glare, the

darker shades of Fieuzal were much more effective than Crookes' glass, and for extreme cases Peacock Blue was the most satisfactory. Artificial light was not stronger in ultra-violet radiation than was sunlight. Dr. Leonard Hill recently stated that in the case of an oil lamp, 97% of the energy was in the infra-red region, and of electric incandescent lamps, 95%. Crookes' A or A2 glass had the superiority that it afforded a general shade without materially disfiguring the appearance or altering color values. There were many people with quite healthy eyes who were, however, physically or mentally abnormal. Often in physical disease and neurasthenia the general function of the eyes was impaired, and in these, help was needed. Smoked glass was still as valuable as ever, but it was somewhat depressing, and Fieuzal did not so much interfere with the color scheme and was as effective as smoked glass in other respects. The wearing of tints was apt to become a habit, and to thus lead to inability to endure bright light unaided.

With regard to wearing tints in disease and as a prophylactic against disease, it had been shown that cataract due to glass-blowing was due to harmful infra-red radiation, not to the effect of ultra-violet rays. The best prophylactic tints were Fieuzal 4 or 5 and Peacock Blue. In all cases of cataract tinted glasses should be worn in bright sunlight or glare. Glare by artificial light should be shunned as much as glare in sunlight. He often ordered two degrees of shade: one for moderate, the other for extreme degrees of light. After the extraction of cataract, the eye was deprived of its most powerful factor of protection against ultra-violet radiation. All aphakic patients should wear some protective tint out of doors, except on dark and cloudy days. In active inflammatory disease or retina and choroid he always ordered Peacock Blue, which cut off all infra-red radiations, and most of the yellow and orange also. It had a very soothing effect on the patient.

Mr. HAROLD GRIMSDALE, in proposing a vote of thanks to the President for his address said he thought a further use of tinted glass was to be found in the case of people towards middle life who manifested Pulfrick's phenomenon. That authority showed, by a series of experiments, that the percep-

tion of a stimulus varied with the intensity of the stimulus, therefore if by any means the stimulus reaching one eye was weakened, as by putting tinted glass in front of it, the position of a moving object was falsely projected. Some people, for this reason, found a difficulty in exactly timing their strokes in sport. Sometimes after retro-bulbar neuritis, although the form perception had been recovered, the light perception had been somewhat reduced, and a moving object was seen in the wrong position.

Mr. ERNEST CLARKE referred to the early electric light with the carbon point, and the glare produced; and mentioned with satisfaction the present practice in many buildings of brightly illuminating a white ceiling, and allowing the hall to be served by a soft diffused light. A great point about Crookes' glass was that it did not cut off luminous rays. He agreed that patients should not be too ready to listen to advertising people on this subject.

Mr. MALCOLM HEPBURN said he had found that the wearing of Peacock Blue glasses on mountain-climbing expeditions was not comfortable; the glasses had to be worn the whole time, whether one was climbing rock or snow, and differentiation was so difficult that a false step was easy. Green was preferable for these conditions.

Mr. G. H. POOLEY (Sheffield) agreed that among the furnacemen the wearing of Peacock Blue glasses seemed a source of great comfort, and they certainly enabled the work to be done efficiently. In the north of England it was found that optical testers were ordering Crookes' glasses in a wholesale fashion, with no such reasons as actuated the medical man, and the public felt that the extra price betokened outstanding merit. Healthy people with normally healthy eyes resorting to tinted glasses was a thing the profession should set themselves to discourage. He did not think it had been substantiated that infra-red rays caused any definite changes in the lens.

Mr. BERNARD CRIDLAND (Wolverhampton) said he understood from the manufacturers of Crookes' glass that the tint had little to do with its optical properties, and that the tint was added because otherwise the public would not think the price was justifiable.

Mr. CYRIL WALKER (Bristol) referred to the success of smoked amber colored glasses in the case of a friend in Labrador; these enabled him to hunt and shoot without discomfort.

Mr. TREACHER COLLINS had a word of praise for amber tinted glasses, following upon his own use of them. When people who came to him were likely to be exposed to ultra-violet rays he ordered them amber-colored glasses. One went to Tibet, and when he came back he said the rest of the party had snow-blindness, but he, having worn the glasses, was immune. He agreed that cooks and others exposed to heat derived relief from wearing Peacock Blue glasses. He would like the President to extend his researches to pebbles.

Mr. A. D. GRIFFITH thought the chief value of Crookes' was in its absorption of the yellow band, by which the luminosity was reduced; this was the center of the diffraction spectrum, and was the region of greatest luminosity.

Cataract Operations in Egypt.

Dr. A. F. MACCALLAN read a paper giving his experiences in ophthalmic practice in Egypt, in which he discussed the various operations, and his own general procedure.

DISCUSSION ON VERTIGO.

Sections of Medicine, Neurology, Ophthalmology, and Otology, Royal Society of Medicine.

By Mr. P. G. Doyne, London.

Sir Humphrey Rolleston opened the discussion from the standpoint of the physician as apart from the otologist and neurologist. He proposed to review the subject from two points of view.—"1. What evidence is there that the action of toxins, transient vascular changes, or functional disturbances of that part of the cerebral cortex, where the sensations concerned in equilibrium normally meet in harmony, may cause vertigo. 2. The ways in which general diseases and visceral disorders affect the vestibular apparatus." With regard to (1) it would seem probable that toxic, vascular or functional disturbance of the cortical end of the vestibular system may occur as instanced by epileptic aura and the occurrence of vertigo in an epileptic and also by the occasional alternation of migraine and vertigo. As disturbances of vision (muscæ volitantes and hemianopsia) may be apparently due to toxic influences or vascular spasm affecting the cerebral cortex so in the same way may vertigo be explained. In this connection it would be interesting to know if deaf mutes with complete labyrinthine destruction experience vertigo when under the influence of vertiginous drugs such as quinine. With regard to (2) it has been suggested that anaphylactic conditions may affect the semicircular canals; cases have been reported of patients with evidence of hypersensitiveness, such as asthma, in whom attacks of dizziness could be produced by taking certain articles of food to which they were hypersensitive. Endocrine disorders have been held

to be able to induce vertigo by altering the vasomotor state of the vestibular labyrinth.

Gastric manifestations and vertigo are more often regarded nowadays as concomitant effects of vestibular disturbance but it would seem reasonable to suppose that toxins arising from faulty action of the liver or intestines might have a direct action upon the vestibular system. In arteriosclerosis the thickened vessels seem more liable to spasm and such spasm affecting the vessels supplying any part of the vestibular system might well cause vertigo. It has been suggested that the giddiness of caisson disease may be due to bubbles of nitrogen gas in the vessels of the labyrinth or cortex.

Laryngeal vertigo may be explained as a result of impulses spreading from the vagus nucleus to the nucleus of the vestibular nerve or as the result of sudden increases of pressure or of small extravasions into the semicircular canals following upon violent coughing. In anæmia it would appear that the cortex is the most probable area affected whereas in erythæmia the vestibular labyrinth. In leukæmia associated with Menière's symptoms infiltration and hæmorrhage of the semicircular canals and in some cases new bone formation has been found at necropsy.

Dr. GORDON HOLMES, speaking as a neurologist, dealt with the various forms of vertigo met with in diseases of the central nervous system.

1. Vertigo due to local cerebral lesions. Considering first lesions in the posterior fossa of the skull. Local lesions anywhere along the intracranial portion of the vestibular tract may cause vertigo. Pressure on a portion of the intracranial portion of the vestibular nerve by a dilated arteriosclerotic vessel being emphasized in this connection. Disease of areas of the central nervous system which are intimately though not directly connected with the labyrinths are a less frequent cause of vertigo. Thrombosis of the posterior inferior cerebellar artery almost invariably gives a history of sudden giddiness. The frequency of vertigo in cerebellar disease is probably overestimated.

With regard to local disease of the fore brain; vertigo occurs when the cortex or subcortical white matter is involved rather than in disease of the basal ganglia or central white matter.

It is very doubtful if a center for equilibrium exists in the cortex. Vertigo may occur from excitation of the centers in the forebrain for movements of the head and eyes. Similar spasmodic movements of the head and eyes may be excited from irritating lesions of the occipital, temporal and parietal lobes.

2. Vertigo with diffuse cerebral lesions. Cerebral arteriosclerosis and in certain cases an increase of intracranial pressure may cause vertigo, though in exactly what way is as yet uncertain. Cerebral concussion and the action of certain poisons must be included in this category. Vertigo associated with these diffuse affections of the brain is usually of a more indefinite type than that associated with local lesions. It is generally referred to as "dizziness."

3. Vertigo as a symptom in other nervous diseases. Epilepsy and migraine are particularly referred to in this connection. Vertigo is often present as an aura or an equivalent in epilepsy. With regard to vertigo as a manifestation of hysteria, it is never safe to regard true vertigo as an hysterical symptom.

Mr. SYDNEY SCOTT, speaking as an aurist, said that septic infection of the labyrinth always caused the symptoms of Menière's disease. The onset of vertigo in the course of otitis media was therefore a symptom of the utmost practical importance, but every case of vertigo occurring in the course of middle ear disease did not necessarily mean invasion of the labyrinth. So long as the patient gave positive caloric tests, it was not necessary to diagnose disease of the membranous labyrinth. In unilateral eighth nerve tumors, vertigo and staggering were constantly present though often not of marked degree. The commonest causes of aural vertigo were not due to labyrinthine disease but arose in connection with Eustachian inefficiency. He would urge that in all cases of vertigo the efficiency of the Eustachian tubes and the posterior nares and the accessory sinuses of the nose should receive particular attention as well as the reaction of the labyrinth to caloric and rotation tests.

Mr. J. HERBERT FISHER, speaking as an ophthalmologist, said that the visual judgments and information obtained from different impulses generated in the extraocular muscles

were elements contributing to the maintenance of equilibrium. Disorders of objective orientation might lead to metamorphosia and errors of judgment but it was doubtful if such ever caused true vertigo. Visual sense seemed to differ from tactile sense, muscular sense and from the sense derived from the semicircular canals in that it was operative when associated with consciousness and was not operative without affecting consciousness. The sense of giddiness produced by gazing at a waterfall was probably due to unperceived ocular movements. The part played by the eyes in the maintenance of equilibrium appeared to depend in the main upon the functions of the internal and external ocular muscles. He was extremely doubtful as to whether astigmatism had any hand in the primary causation of vertigo though in such cases accurate correction of the refraction was undoubtedly advisable. Train sickness and seasickness were probably in part at least due to disturbance of the ocular muscles when viewing moving waves or objects. Heterophoria was probably of little importance with regard to vertigo. In tabes the eyes can maintain the equilibrium of the body as evidence by Rhombergism, and in states of alcoholism when giddiness is experienced in the dark it can be eliminated by switching on the light and bringing the eyes into play. In conclusion he would suggest that the sense of vision contributes to equilibrium only when it affects consciousness through the higher centers. The sense of sight and the information from the ocular muscles are not essential for the maintenance of equilibrium. Normal visual impulses normally projected on the sensorium may contribute to equilibrium and may control a vertigo generated by disorder of other mechanisms, but if abnormally transmitted to the sensorium may produce a sense of vertigo.

Sir JAMES DUNDAS GRANT said that small doses of quinine had so great an effect in checking attacks of aural vertigo that in a unilateral case if quinine did not stop the vertigo it was probably not due to a semicircular canal condition. He agreed as to the important part that disease of the middle ear played in cases of Menière symptoms. He believed that this was due to the movements of the stapes and of the membrane of the foramen ovale which normally acted as a safety

valve to sudden increases of vascular pressure in the internal ear but which became fixed after middle ear disease and could no longer act in this way. Concussion was a frequent cause of vertigo. He had found that great relief was afforded in these cases by small doses of perchloride of mercury.

DISCUSSION ON "THE COMPARATIVE VALUE OF COCAINE SUBSTITUTES."

ROYAL SOCIETY OF MEDICINE, SECTION OF LARYNGOLOGY, MARCH, 1924.

BY MR. P. G. DOYNE, LONDON.

Dr. P. WATSON-WILLIAMS said that recently much investigation had taken place to find a satisfactory substitute for cocaine. The toxicity of cocaine and its abuse by the narcomaniac had brought the matter much into the public eye. With regard to the question of drug addiction, he thought this overstressed. It certainly was desirable to limit the use of cocaine strictly for surgical purposes. Various substitutes which were said on their introduction not to produce the addiction habit later turned out to be almost as bad as cocaine in this way. In considering the choice of a local anæsthetic the exact type of anæsthesia required must be considered—the site area and nature of the lining membrane, and the choice between surface absorption, infiltration or nerve blocking. He proceeded to consider the question from the standpoint of the laryngologist.

Eucaine (5–12 per cent. eucaine lactate in aqueous solution) he found fairly efficient for anæsthesia but though non-irritating it did not afford good ischæmia. Butyn (2–5 per cent. solution) he found had a stronger anæsthetic effect than eucaine but it was considerably more toxic and further has to be associated with adrenalin if ischæmia of the tissues is desired. He thought that it was fallacious to consider these cocaine substitutes safe unless the dosage was very carefully estimated. It was to be remembered that the use of cocaine was far more widespread and in consequence mortality statis-

tic for it would be proportionately high. He himself looking back over many years could recall only three cases of serious fainting following the use of strong cocaine applications and no case of drug addiction. For routine examination of the nose and for minor proceedings he used 2–10 per cent. aqueous solution of cocaine. For exploration of nasal sinus she applied 20 per cent. cocaine with $\frac{1}{20000}$ adrenalin followed by solid cocaine crystals in adrenalin $\frac{1}{10000}$ cautiously applied by a cottonwool probe, 3 grains of cocaine crystals being the full dose never exceeded.

Mr. R. FOSTER MOORE pointed out that those local anæsthetics were used in different ways by the various specialties. It would be interesting to find out if addiction to any of the cocaine substitutes was established as there were no penalties attached to the possession of them. It was desirable to know the properties of the substitutes when used in the solid form as compared with cocaine. The fact that cocaine had an action on the pupil, causing dilatation, was on the whole an advantage to the ophthalmic surgeon. He suggested the possibility that the action of these drugs on the sympathetic nerve might be substantiated as a test of the power of the drugs to act as a stimulant to the central nervous system and so be likely to become addiction drugs. Speaking as an ophthalmic surgeon for infiltration anæsthesia he was entirely satisfied with novocaine 2 per cent., but for intraocular operations cocaine so far led the field.

Mr. F. N. DOUBLEDAY speaking as a dental surgeon pronounced in favor of novocaine 1½ per cent. as the most satisfactory anæsthetic for dental purposes.

Dr. BLOMFIELD speaking as an anæsthetist said that he used novocaine 2 per cent. almost entirely for local injection. For spinal injection he used stovaine. The chief danger of stovaine was the lowering of blood pressure which it produced. This, however, could be effectively guarded against by the head down position of the patient soon after the drug had been introduced.

Sir WILLIAM MILLIGAN said that for aural work on the whole he preferred cocaine. Butyn did not cause contraction of the tissues which was a great disadvantage in a confined space like the ear, as also was its tendency to induce hyper-

æmia. Its action was, however, rather more rapid and of longer duration than cocaine. He knew of 2 patients who had contracted the cocaine habit after the use of the nasal spray.

Mr. E. WATSON WILLIAMS said that experiments on monkeys had shown that butyn was at least as toxic as cocaine. He pointed out that the hyperæmia associated with butyn caused rapid absorption of this drug.

Mr. F. ST. J. STEADMAN used novocaine 2 per cent. for injection of the inferior dental nerve in the pterygoid-mandibular fossa prior to tooth extraction. He found that with butyn used in this way there was more after pain than with novocaine.

Dr. W. HILL preferred cocaine for nasal application and novocaine for infiltration anæsthesia. Butyn he found uncertain in its action as an anæsthetic and eucaine was also uncertain in this respect. With stovaine he had seen toxic symptoms.

Dr. ALBERT GRAY preferred cocaine for aural work. He combined it with suprarenal extract. This caused slow absorption of the cocaine and he thought this an important factor in preventing toxic symptoms.

Mr. T. B. LAYTON said that he was not always able to distinguish between the toxic symptoms of cocaine and its substitutes and the symptoms seen in mental states engendered by fear and apprehension. He thought that the confidence with which the surgeon applied the anæsthetic was a factor to be taken into account. He referred to the lack of hæmostasis which was associated with butyn.

Mr. MARK HOVELL said that he had given up using cocaine for general nasal work. He used eucaine (5–10 per cent.) solutions, and for clearing a nostril of polypi novocaine 2 per cent.

Dr. HORGAN said he had used alypin for surface anæsthesia in the nose but he had found it more toxic than cocaine. With regard to counteracting cocaine poisoning he stressed the advantage of giving a hyperdermic injection of hyoscine twenty minutes before the operation and the value of giving an intravenous injection of 10 per cent. solution of calcium chloride in severe cases of cocaine poisoning.

Mr. SYDNEY SCOTT said that he had found butyn useful in some cases that did not tolerate cocaine well but he did not think it would displace cocaine. He had never known of a case of addiction.

Mr. HERBERT TILLEY had found novocaine entirely satisfactory for resection of the nasal septum and for nasal operations on the antrum, he had also used it successfully for tracheotomy where a general anæsthetic was contra-indicated. He did not think that there was yet a safe local anæsthetic which combined good anæsthesia with contraction of the tissues, a cocaine substitute must combine both these qualities satisfactorily in order to displace cocaine.

Mr. P. WATSON WILLIAMS, in reply, said that for the profession to taboo cocaine only on account of its social abuse before effective restrictions had been imposed on its sale to the public was the wrong attitude to adopt.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. DAVID H. WEBSTER, Secretary.

MEETING OF MAY 19, 1924. DR. BEN WITT KEY, CHAIRMAN.

Dr. John M. Wheeler presented a case of **Bilateral Ectropion of the Lower Eyelids** due to lupus.

The face and the lower lids were severely involved, and a cicatricial tissue process resulted which everted the lower lids and pulled the tissues about the eyes well down out of place. Large true skin grafts were taken from the upper lids in such a way as to correct the ectropion and lift the outer canthi. Two intermarginal adhesions were made on each side. The sutures were left long and attached to the forehead so as to support the lower lids and outer canthi. The operations were performed 19 days ago. Both grafts were complete takes and there is a correction of the deformities, which will be permanent.

Dr. Wheeler said that in other countries most surgeons had failed to get complete takes with free grafts of true skin and some considered it impossible; that a high percentage of successes could not be obtained unless the surgeon understood the proper technique.

Discussion: Dr. Weidler: Six months ago Dr. Wheeler came to the Manhattan Eye, Ear and Throat Hospital and operated on a case of burn. The night following the woman became delirious and repeatedly tore the bandages off, yet the graft took completely. At the present time the result is perfect.

Dr. Wolff: How long does it take to perform the operation?

Dr. WHEELER: First—I do not touch the graft with forceps in removing it nor do I use forceps and artery clamps in the bed where the graft is to be placed. Second—Secure intermarginal adhesions are extremely important, and should be left for a period of months after operation. Third—The matter of pressure is very important and there is no danger of this being too severe. First gutta percha tissue with a very thin smear of vaseline is laid over the graft. This is put on in two layers with the graining crossed. Gauze is firmly secured by adhesive, then a pressure bandage is applied, then more adhesive with very severe pressure over the bandage. There is no danger of injury to the cornea, as the intermarginal sutures hold the lids margins in close apposition. Fourth—After the dressing is put on it should be left in place for five days. Fifth—The graft should not be allowed to become dry after the operation, but should be kept covered with gutta-percha tissue for about eight weeks and then vaseline should be applied with gentle massage daily for another month.

Dr. S. GREEN presented a case of **Coloboma of Optic Nerve.**

DISCUSSION: Dr. TYSON: I saw a similar case with coloboma of iris, optic nerve and retina.

Dr. JULIUS WOLFF presented a case of **Traumatic Cyst of Iris with glaucoma.** Patient aged 32.

In 1912 he sustained a perforating injury of the left eye, which resulted in cataract formation, and several years later the formation of an implantation iris cyst. In 1914 Dr. Carl Koller operated for removal of the cyst, which was situated in the lower, outer quadrant. The lens at that time was opaque. In 1919 the tension of the eye had risen and Dr. Koller performed an iridectomy above. The lens at that time was shrunken, but the cyst had not yet recurred. Over a year ago the cyst recurred and tension at times was elevated. When seen by Dr. Wolff in March of this year, the cyst filled at least three-fifths of the anterior chamber, and was adherent to the cornea as well as to the thickened secondary membrane and the remains of the iris. The tension was over 50 and the eye painful. Healing was uneventful, and at the present time the cyst is about one-third as large as before operation, and the eye thus far has remained soft. The patient can count fingers at three feet, and the field is good. If the eye remains soft a

later attempt at improvement of vision can be made by dividing the capsular membrane.

Dr. MARTIN COHEN presented a case of **Orbital Tumor (Neuro-Myxoma)** which was operated upon by the Krönlein method (to be published).

DISCUSSION: Dr. WEIDLER: In doing the Krönlein, did you use a saw or a chisel?

Dr. COHEN: A chisel.

Dr. ROSE: How long did it take?

Dr. COHEN: Two hours.

Dr. WEIDLER: I think that we do not do this operation as frequently as we should, instead of doing an exenteration.

Dr. COHEN: The condition of the optic nerve is due to secondary optic nerve atrophy, while the injury of the muscles is probably traumatic.

Dr. KEY: The arteries seem very small.

Dr. COHEN: The appearance of the arteries is probably within physiological limits.

Dr. ARTHUR TENNER demonstrated a **Lachrymal Needle** with the following features:

1. A conical (solid) tip.
2. Small lateral opening.
3. Conical shape.
4. A slip-on needle, that can be attached to any syringe.
   Advantages:
   (a) Small solid tip.
   (b) Conical shape of a dilator, by this means sac can be irrigated, anesthetized and dilated at the same time.
5. Needle made of non-corrosive material.

DISCUSSION: Dr. KING: Would it be an advantage to have it curved?

Dr. TENNER: No.

Dr. BONN: Is there any advantage for an additional opening?

Dr. TENNER: I have found this very satisfactory.

Dr. KEY: For twelve years I have used a small gold needle, which I have made blunt. In using a pointed needle in the punctum, it passes without pressure.

Dr. KING: I mentioned the curved needle as I have found

it easy to turn the tip down and irrigate the sac into the nose.

Dr. WALTER WEIDLER presented a case showing an **Optico-Ciliary Vessel.**

Dr. KING saw one case like this, but the vessel reappeared some distance from the disc.

Dr. JOSEPH SHELDON SOMBERG presented a case of **Unilateral Hemorrhagic Retinitis.**

Patient, age 64, had failing vision for six weeks in his left eye. One month ago retinal vessels were contracted and the whole retina was covered with minute hemorrhagic spots, none any larger than the head of a pin. There was also a slight swelling of the disk. General examination was entirely negative. Blood pressure $\frac{140}{80}$. The right eye at no time showed any involvement.

At the present time the hemorrhages are slightly larger, but of uniform size, and there is no change in the condition of the optic disk.

DISCUSSION: Dr. HOMER SMITH: Has a blood chemistry been done?

Dr. SOMBERG: Yes. The blood chemistry showed no abnormalities with the exception of a very slight increase of the blood urea which may be considered within normal limits.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M. D., F. A. C. S., New Rochelle, N. Y.

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29. VILLARD. **Ocular complications in pneumonia and broncho-pneumonia.** *Annales d'oculistique*, clix., p. 746.

30. VOGT, A. **Copper cataract with chalkosis retinæ.** Meeting of Swiss ophthalmologists, Schaffhausen, June 24 and 25, 1922.

31. WISSMANN. **Diseases of the eye in pregnancy, with especial reference to eclampsia.** German ophthalmic society, Jena, June, 1922.

32. YUDKIN, A. M. **Ocular manifestations of the rat which result from deficiency of vitamin A in the diet.** *Journal A. M. A.*, Dec. 30, 1922.

FERNANDO (11, **The eye in beri-beri**) describes three cases which showed slight defects in vision, photophobia, night blindness, slightly contracted pupils, narrowing of the field for green and slight hyperemia of the disks. The improvement was slow following a change of diet.

FRANKLIN and CORDES (12, **Lupus vulgaris with ocular extension**) describe a case of lupus vulgaris which involved the greater part of the face. The right eyelids were markedly contracted from cicatrices leaving the cornea exposed. The cornea was opaque and dry. At the limbus were a number of ulcerations and nodules which proved on examination to be

tuberculous. A low grade inflammatory process was present in the conjunctiva and ciliary body. The corneal epithelium had become epidermoid in type which happens after long exposure.

According to ADIE (1, **Dystrophia myotonica (myotonic atrophica), an heredo-familial disease with cataract**) this disease appears to be much more common than is usually supposed and would be more frequently recognised if careful watch is kept in outpatient clinics. The disease must now be regarded as a general disease with widespread symptoms and manifestations, the muscular symptoms being only a part of the whole. The onset of the disease is most frequent between the ages of 20 and 30 and males are more often attacked than females in the proportion of 5 to 1. The symptoms fall roughly into 4 groups:—1. Muscular. 2. General dystrophic symptoms. 3. The so-called tabetic symptoms (loss of tendon reflexes, etc.). 4. Other symptoms (the speech defect, stigmata of degeneration, psychical defects, etc.). Concerning the muscular atrophy; it usually appears first in the following groups in the following order—head and neck, arms, legs—different muscles in these groups may be affected unequally and there is no exact rule of precedence but absence of some weakness in closing the lids is rarely absent. In some advanced cases every muscle in the body is wasted. Myotonia is shown by delayed relaxation of muscular contractions produced voluntarily or by mechanical stimulation, is confined to or most conspicuous in the hand-grasp. Myotonia and wasting do not by any means go hand in hand, the same muscle not being affected by each condition to an equal degree. Of the extra-muscular symptoms cataract is of great interest. It usually begins as a posterior cortical cataract and ripens quickly. The great importance of cataract in this condition lies in the fact that it occurs in otherwise healthy members of the dystrophic generation and is common in healthy members of the preceding generations. Further the appearance of the cataract shows the phenomenon of "anticipation" in succeeding generations. The disease is familial and hereditary in every instance but it may appear not to be so unless careful enquiry is made as isolated cases often occur in succeeding generations, further the disease may skip several generations

and then break out again but it would seem on the whole to eventually die out. Of the extra-muscular symptoms, atrophy of the testicles, baldness, loss of body weight, and increased secretion of sweat and tears are the most frequent. Loss of tendon reflexes is a common sign and is probably secondary to changes in the muscles themselves. On the whole overmuch attention has been paid to the psychic changes. In considering the ætiology of the disease it would appear that many of the symptoms might well be explained by disorders of the endocrine system, but on the whole Adie thinks that it would be premature to classify the disease as a polyglandular syndrome.

P. G. DOYNE.

ARLT (2, **Hemianopsia in encephalitis lethargica**) reports the case of a patient who suffered sevcre pain in the right side of the head for several weeks during an attack of encephalitis. Investigation of the visual fields revealed a complete left hemianopsia, sparing the macula on the left side to about $1\frac{1}{2}°$, on the right to 5°. In the affected parts of the field the patient saw gray stars. Nothing wrong otherwise could be found in either eye. The case is noteworthy because the hemianopsia appeared to be the only symptom, and this proved transitory, complete recovery following.

In the 10 cases reported by CAMERON (4, **Clinical aspects of eye symptoms in encephalitis lethargica**) there was paralysis of the oculomotor nerve in 8 and of the 6th in 2 cases. There were 2 cases of paralysis of the 7th. In 5 there was some degree of optic neuritis. In all but one case there was paralysis of accommodation and in some dilatation of the pupil. The loss of accommodation seems to be among the first symptoms to appear and the last to recover.

In the case reported by JESS (19, **Cortical blindness after encephalitis lethargica**) there was at first cortical amaurosis, and then the left lower quadrants of the visual field began to become restored, the improvement continuing until colors could be perceived. The left upper quadrants and a small zone in the right lower quadrant at the middle line remained color blind. After this improvement during convalescence followed a progressive deterioration in the function of the cortical retina, such that at the end of a year gross movements

could be perceived in only a very small area of the lower quadrant and colors could not be perceived at all. The patient was practically blind.

SPILLER (26, **High grade choked disks in epidemic encephalitis**) reports two cases which are of interest because choked disk is a very rare accompaniment of this disease.

BAB (3, **Ocular changes in lymphatic leucæmia**) reports the case of a patient 57 years old who was under medical treatment for a leucæmic lymphadenosis before the blood picture showed any change. The right eye presented a plastic iritis with glaucoma, the formation of an exudate in the anterior chamber, and vitreous opacity. Iridectomy was performed twice. The exudate in the anterior chamber removed at the time of these operations proved to be composed of lymphocytes. In spite of treatment the glandular swellings became generalized and the patient died some weeks after an increase of the white blood corpuscles had appeared in the blood picture.

CLAUSEN'S (8, **Uncommon fundus changes in myeloid leucæmia**) patient was a man 39 years old. The blood examination showed 3 millions erythrocytes, hæmoglobin content 65%; 682,000 leucocytes; neutrophile myelocytes 23%, eosinophile 15%, basophile 11%, lymphocytes 0. Each fundus showed a great distention and tortuosity of the veins. The fundus was only slightly yellow, with a little opacity of the retina. In the periphery, in the region of the equator, were very large gray infiltrations which protruded like a tumor 2 or 3*mm* into the vitreous, with a broad red edge at the base and slight hemorrhages at the top. These were present in both eyes and thought to be retinal lymphomata. Aside from a broad, grayish white sheath of a vein in the left eye, the vessels showed no other changes.

GOSS (15, **Effect of blood transfusion on the retinitis of pernicious anæmia**) concludes from his studies that transfusion does not prevent the further occurrence of hemorrhage of the retina nor does it cause the hemorrhage to absorb more rapidly. The remote effect is a gradual lessening of the retinal œdema and decreased tendency to hemorrhage.

GALLUS (14, **Eyes of diabetic war veterans**) studied 76 cases, 47 of the patients between the ages of 26 and 40 years.

In 42 the eyes were normal in every respect, in 34 pathological conditions were present. Retinitis was found in only one patient, a man 62 years old, which goes to support the theory that retinitis is an accompanying symptom of diabetes only in old age, when the disease has already existed long. The almost total absence of lenticular opacities, even after very careful examination with dilated pupils, is striking, a cataract being found in only one patient, a man 24 years old with severe diabetes. Gallus believes that other factors, probably hereditary, must act with the diabetes in the production of cataract, and remarks that diabetic Jews are particularly prone to cataract. Retrobulbar neuritis was present in fourteen cases, in nine of whom abuse of alcohol and tobacco could be excluded with certainty.

LUEDDE'S (22, **The significance of the tuberculin reaction and other problems in ocular tuberculosis**) paper is a fair minded discussion of views now held regarding the diagnostic and therapeutic uses of tuberculin. He lays stress upon the importance of eye lesions associated with diseases of the nasopharynx, suggesting that the processes in the nose may be of tuberculous nature and carried to the eye through the lymph channels. Treatment of the nose therefore in his estimation is very important. By means of a questionnaire sent to representative pathologists and bacteriologists he attempted to obtain their views as to whether they regard focal reaction of tuberculin as specific and whether focal reactions have been observed following injections of non-specific substances as for example milk. The answers were varied. He concludes that a focal reaction from tuberculin makes the diagnosis highly probable and that it can be explained either as a specific or non-specific effect.

VILLARD (29, **Ocular complications in pneumonia and bronchopneumonia**) divides these complications into two groups, extrabulbar and intrabulbar. The less common of the former affect the conjunctiva, optic nerve, and motor nerves of the eye, while those affecting the cornea are observed more frequently. Keratitis may occur in the course of a pneumonia, but is more common as a complication of a severe bronchopneumonia, especially when it follows German measles, usually appearing as a keratitis with hypopyon, inter-

stitial keratitis, or keratomalacia. The prognosis is grave, for the sight of the affected eye is commonly lost. The theory of an exogenous infection, from the hands or cloths soiled with pneumonic sputum, has much in its favor, particularly when the power of resistance of the patient has been reduced by the pulmonary disease. Among the intrabulbar complications are diseases of the iris, of the retina, of the entire contents of the eyeball, and of the deep membranes. The affection of the iris may appear as a paresis of the accommodation, or as an iritis, but most frequently appears as an anisocoria with the larger pupil on the side with the inflammation. Inflammation of the retina presents the picture of septic retinitis which closely resembles that of retinal tuberculosis, but is of a benign nature and always recovers. The pathological processes in the entire contents of the eyeball and in the deep membranes are extremely harmful, forming the so-called metastatic ophthalmia. This appears as a plastic and as a purulent iridochoroiditis, the latter just as serious as panophthalmitis. Its origin is endogenous, the consequence of emboli of pneumococci in the vessels of the retina, or of the uveal tract.

DUVERGER and BARRE (9, **Ocular troubles in Parkinson's disease**) report the eye symptoms in six cases, part of them cases of true paralysis agitans, part of them cases of the pseudo variety following encephalitis. Ptosis and blepharochalasis occurred in only one eye in the true cases, but were often bilateral in the pseudo cases, though without enlargement of the lid skin. Twitchings of the orbicularis were frequent. Absence or weakening of the convergence was noted with a corresponding diplopia. Nystagmus when the eyes were turned far to the side was observed, combined with a labyrinthine disturbance.

CIRINCIONE (7, **Ocular lesions in affections of the sella turcica**) concludes as the result of a long series of studies with the aid of X-ray pictures that about ten per cent. of all atrophies of the optic nerve are occasioned by affections in the sella, or in its neighborhood. He designates as pathognomonic the fluctuating condition of the vision and of the borders of the visual field which is most marked shortly before the final blindness while the atrophic papilla appears to be unchanged. This does not necessarily mean that a tumor of the hypophysis

is present; sometimes there is a cyst containing blood and serum at the sella turcica, sometimes a hypertrophy of the hypophysis, more rarely a hydrocephalic dilatation of the third ventricle which presses upon the chiasm and may lead to a cystic transformation of the entire infundibulum. The course of diseases at the sella may extend over many years and hypophyseal symptoms, in addition to the optic atrophy, may be wholly absent. Cirincione questions the advisability of an operation when a tumor is present, and relies rather on irradiation. The prognosis of an operation he considers good only in cysts of the sella, glandular, hypertrophy, and ectasia of the infundibulum caused by hydrocephalus internus of the third ventricle.

HIRSCH (17, **Radium treatment of hypophyseal tumor**) reports the case of a patient 30 years old from whom a portion of a solid tumor of epithelial origin was removed by operation. Improvement followed, but at the end of nine months the visual fields began to contract. Hirsch then applied radium in several sittings. At first these were 27 hours, after four months ten hours long, with 18*mg* of radium. This treatment resulted in the restoration of an almost normal field for the right eye, and a distinct improvement in that of the left. He prefers radium to X-ray treatment because of the danger that the latter will cause serious anæmia and cachexia. It is essential that the tumor be laid bare so that the radium may act directly upon it without passage of the rays through other tissues.

LILLIE (21, **Ocular phenomena produced by intracranial lesions involving optic tracts near the chiasm**) in sixty-two cases of proved chiasmal lesion found that the fields all followed the same general type and that the defect always involved the blind spot. He lays great stress on the importance of the color field. The origin of the lesion affecting the chiasm cannot be definitely determined by ophthalmoscopic examination. ALLING.

MARZIO (23, **Chiasmatic syndrome in affections of the hypophysis**) investigated six hundred cases of simple optic atrophy and found changes of the hypophysis to be the cause in sixty. Ten of these cases were associated with acromegaly, two with

acromegalic hypertrophy, one with infantilismus, four with habitus adiposo-genitalis; in all the others the visual disturbances, varying in degree, formed the only symptom. The diagnosis was confirmed in each case by the X-ray picture. Although the visual disturbances form the only constant symtom, these may be so slightly marked, and, like the tumor of the hypophysis under certain circumstances, so little progressive, that they may not become subjectively noticeable for a long time, and possibly may remain latent throughout life. Marzio ascribes to this the relative rarity with which tumors of the hypophysis are observed clinically, and points out that when such tumors are clinically observed they are almost always far advanced. The first of the chiasmatic symptoms is a descending optic atrophy. This is almost constant and goes hand in hand with the change in the visual field. Choked disk was found in five cases caused by hydrocephalic distention of the third ventricle, but only one case of optic neuritis. In 50% bitemporal hemianopsia was present in its characteristic form. At the beginning of the disease as well as toward its end disturbances in the visual fields are very frequent which present pictures that vary a great deal in correspondence with the direction of growth of the tumor. Twice he found homonymous, twice binasal hemianopsia, the latter always in conjunction with central scotoma. A slight bilateral exophthalmos existed in two-thirds of the cases with acromegaly. Pareses of the nerve to the extrinsic muscles were rare, the fifth and third seemed to be the soonest affected. Characteristic of hypophyseal affections is the fluctuating condition of the vision, which varies at different examinations in correspondence with the fluctuating pressure of the tumor.

Van Lint (28, **Syndrome adiposo-genital**) reports two cases. In one autopsy revealed a tuberculous tumor of the meninges which pressed upon the chiasm. As the operative removal of such tumors is attended by very great danger to life he recommends that the X-ray or radium should always be tried first.

Levison and Alter (20, **Glioma of the optic thalamus**) present the history of a man, 66, who developed dimness of vision and pain on the right side with the right eye showing the picture of hemorrhagic retinitis. Later on choked disk and proptosis developed rather suddenly in the right and a punctate

retinitis in the left. The right field showed contraction on the nasal side. There was no paralysis, ataxia or sensory changes. At autopsy a tumor was found involving the thalamus region and impinging into the third ventricle. It was the size of an English walnut and proved to be a pure glioma.

ROBERTS (25, **Chloroma**) states that ninety cases of chloroma have been reported in literature. This is a malignant disease akin to sarcoma, characterized by blood changes and the occurrence of pale green tumors throughout the body. His case was that of a boy, thirty-two months old, who first developed exophthalmos and died of multiple tumors of the body. The histological examination showed that the tumors consisted of small round cells closely packed with a connective tissue stroma. Mononuclear eosinophiles were also found.

ALLING.

In order to prove the incidence of this phenomenon CARVILL (6, **Bitemporal contraction of the fields of vision in pregnancy**) examined one hundred pregnant women and found only five who could be said to have normal fields of vision. The temporal contraction was most evident as the time of parturition approached. Of the fifty-one cases examined postpartum all but two showed complete recovery. The cause of these defects in the field is a compression of the chiasm produced by the physiological enlargement of the hypophysis during pregnancy. ALLING.

WISSMANN (31, **Diseases of the eye in pregnancy, with especial reference to eclampsia**) found in over ninety per cent. of his cases a blood pressure of 180 and upwards. Neuroretinitis was the most common form of disease. Amaurosis was the only trouble which he met with in all conditions of the blood pressure, the lowest being 140. A small number of cases of neuroretinitis occurred with the blood pressure below 180, the lowest being 160, but in the great majority it was over 180. Detachment of the retina was met with only when the blood pressure was over 200. Œdema of the papilla and of the adjoining retina he is inclined to class with amaurosis rather than with neuroretinitis.

TERRIEN (27, **Post hemorrhagic amaurosis**) reports two cases of eye trouble after severe hemorrhage. The first case

was that of a 30 years old woman who had a profuse hemorrhage after delivery. Four months later there was a partial atrophy of both papillæ with a marked contraction of the lower halves of the visual fields. The second case was that of a soldier 24 years old, whose left leg had to be amputated after he had suffered a severe loss of blood. Some weeks later optic atrophy set in, with a marked contraction of the lower portion of the visual field. Terrien believes that in these cases the harm is done by the draining out of the minute arteries in the retina and optic nerve increased by a spasm of the vasoconstrictors induced by a lack of oxygen in the blood.

CANDIAN (5, **Septic embolism of the retina following extraction of a carious tooth**) reports a curious case. A patient 40 years old had a tooth extracted. Immediately afterwards a high septic fever set in. Three days later the patient noticed loss of vision, and there was to be seen in the region of the macula a large, elevated white patch with a network of little retinal vessels at its margin and some hemorrhages on its surface. Some days later iritis with hypopyon appeared. Then another carious tooth was extracted, after which the hypopyon cleared up. Then a white exudate could be seen in the macula, bordered by an œdematous gray zone, and with a white cord extending from it floating in the vitreous. The vision was fingers at a meter. The course and final result were unusual; as the inflammatory symptoms passed off, the vitreous opacities cleared up, and the eyeball as such remained uninjured, while usually all septic emboli in the eye lead to panophthalmitis.

GUTZEIT (16, **Blue sclera and fragility of bones**) observed a case of this nature in a 19 years old girl. The eyes presented no special condition aside from the gray blue scleræ. The abnormal fragility of the bones had led to more than twenty fractures. The ligaments and capsules were capable of abnormal stretching. Other abnormal conditions were absence of the thyroid, relative lymphocytosis, early loss of teeth, and a markedly hydrocephalic rachitic cranium with a flat sella turcica. Contrary to the findings in most of the published cases no hereditary predisposition could be determined. Gutzeit thinks that the rickets and the disturbances of the internal secretions were answerable for the condition.

YUDKIN (32, **Ocular manifestations of the rat which result**

from deficiency of vitamin A in the diet) fed rats on diet free from vitamin A and observed first lacrimation and photophobia. Later the eyelids became œdematous and the lacrimal secretion became viscid. Fat-like yellowish white patches of secretion soon appeared in the fornices. As the process continued plaques of secretion were found on the cornea and then ulcers which finally developed into panophthalmitis. Marked degenerative or inflammatory changes were noticed in the lacrimal gland. He thinks that these lesions are similar to those observed in man in xerosis and keratomalacia.

EVANS' (10, **Quinine amblyopia**) patient while under the influence of alcohol took about 100 grains of quinine sulphate and a few hours later his vision was reduced to light perception. He had no nausea, vomiting, deafness, tinnitus or diarrhea. The fundi showed pale nerves, small arteries and enlarged veins. There were plaque-like spots of exudate along the course of the arteries. The fields were reduced to very narrow limits.

The GAINSBOROUGHS (13, **Quinine amblyopia**) report the case of a man 41 years old who took about sixty grams of quinine in tablet form at a single dose. Three hours later his sight was reduced to perception of light, his pupils dilated and not responsive to light, the fundus was hazy, the papillæ pale, and the arteries very small. Improvement quickly followed the inhalation of amyl nitrite.

JESS (18, **Gas injuries of the eyes**) says that only in certain unfortunate cases in which the concentrated gas was squirted directly into the eye was there a severe burn of the cornea which might cause the loss of the eye. Commonly gas causes only a transient irritation of the conjunctiva, often with great lacrimation and severe photophobia which renders the victim unfit for military duty for a short time, but without serious injury. This is true of what is commonly known as tear gas. Other gases used during the war are mentioned which cause severe irritation of the eyes often with injury to the cornea. Occasionally hemorrhages into the retina and vitreous were observed, also thrombosis and embolism of the central arteries, and post neuritic optic atrophy. Hemeralopic troubles frequently persist after the clearing up of slight chorioretinitic injuries. But intraocular troubles were comparatively rare in

the large number of soldiers who were gassed. The severest irritation of the external eye was produced by the so-called yellow cross gas, dichlorethylsulphide. The bursting of shells scatters about the petroleum-like fluid with which they are filled, from which arises a widely distributed gas. After a period of latency lasting six or eight hours there appears an enormous swelling of the lids associated with blebs, and a great œdema of the conjunctiva with whitish burns of the conjunctiva and cornea in the region of the palpebral fissure. These symptoms are accompanied by severe pain and blepharospasm. In spite of the very threatening picture the prognosis is good under correct treatment. Only in rare cases was there secondary suppuration of the cornea and loss of the eye. Soothing treatment is indicated, such as is furnished by alkaline ointments. The severest injuries heal in a few weeks leaving more or less dense opacities of the cornea, but there often remains a marked irritability of the conjunctiva with a tendency to the formation of superficial corneal infiltrates. Disturbances within the eye seems to have not been caused by this gas.

Reis (24, **Disease of the optic nerve caused by trinitrotoluol**) describes two cases. The first was that of a munitions worker who had filled shells for four years. In 1918 visual disturbances appeared which were ascribed at first to a fall upon the back of the head. A careful examination in 1919 revealed a great reduction of the central vision with an absolute central scotoma for green and red. No fundus changes were noted until a year later, when a temporal pallor of both papillæ and very small retinal arteries were observed. The second case was that of a workman who had withdrawn trinitrotoluol from casks. In this there occurred a very rapid loss of vision, such that when he awakened he was blind. No other signs of poisoning were present. After a few days there was a return of light perception, followed by improvement until vision was $\frac{1}{2}$. At the first examination on the day after the poisoning occurred there was a marked cloudiness and redness of the papilla with a complete absence of light perception. Six months later the papillæ showed a marked temporal pallor with sharp margins.

Vogt (30, **Copper cataract with chalkosis retinæ**) reports

two cases. The first patient, a man 23 years old, presented a yellow, shining reflex in the macula, which looked as though a source of light was situated in the macula. In the lens was found a bit of copper. Questioning elicited that some years before something struck his right eye while he was shooting, that the eye was a little irritated, and that it was examined by a physician. Disturbance of the vision did not appear until long after the injury. Vogt terms the reflex from the retina chalkosis retinæ. The retinal vessels showed yellowish sand-like reflexes, and irregularly round yellow patches surrounded the macula in a circle, as in retinitis circinata. In the lower segment of the posterior surface of the cornea there was a greenish discoloration of the endothelium. In the vitreous there was a snow white framework of threads with dense, gray-green masses of points interspersed. A very small bit of copper was located accurately by means of the X-ray and was removed through an incision in the sclera without distinct loss of vitreous. Two months after an uneventful recovery detachment of the retina took place.

(*To be continued.*)

BOOK REVIEWS.

I.—**Eyeless Sight.** A Study of Extra-retinal Vision and the Paraoptic Sense. By JULES ROMAINS (Louis Farigoule). Translated by K. C. OGDEN, 251 pages, 1924. G. P. Putnam's Sons, New York.

Dr. Romains has conceived the idea that the skin has retained its supposedly primitive function of reacting to light, and that this function may be cultivated to such a degree that the skin may become a useful organ of sight. He bases his theory on the biologic fact that in our earliest progenitors the outer covering of the body, or "skin" performed all the sensory functions of the body—reacting alike to light, heat, sound and touch, and therefore by a stretch of imagination these forms could be said to see with their skins.

Other primitive functions have been, in a measure, retained even after special organs have been developed for carrying on such functions. For example, at first the outer covering of the body was its only excretory organ; later kidneys were developed to carry out this function, but the skin still retains sweat glands, which, even in the highest forms of life still function as excretory organs. So with respiration, the skin is supposed to retain to a slight degree its respiratory function though lungs have been developed for that purpose.

Dr. Romains believes that the oft reported phenomenon of somnambulists walking with their eyes shut and easily avoiding obstacles in their way, and the claim by certain persons known to be blind that they can see, may be taken as evidence that in these cases, at least, the skin is functioning in the place of the eyes.

He thinks also that we possess anatomic structures capable of carrying out this function, and points to certain of the cor-

puscles of Ranvier in the epidermis, which are crudely formed bi-convex disks the inner surfaces of which are in contact with afferent nerve terminals. He thinks that these bodies may actually form minute images, and that their afferent nerves act as optic nerves, transmitting visual impressions to the brain where they are interpreted much in the same way that we interpret ordinary visual impressions.

Dr. Romains then proceeds to the description of numerous experiments that he has performed in an effort to prove his theory, many of which were performed in the presence of apparently competent witnesses. The results of these experiments—if they can be credited—are almost beyond belief. His subjects, whom he places in a certain stage of hypnosis, with their eyes securely blind-folded, are made to recognize and describe numerous objects placed within a few feet of exposed portions of the skin, and later, when better trained, to read pages from books or newspapers.

If there is any scientific basis for Dr. Romains' claims, his experiments will be quickly verified by others. Until then it must be borne in mind that these claims offer an opportunity to dishonest persons to take advantage of the unfortunate blind by claiming the ability to teach them to see with their skins. A. H. T.

II.—**Grundzüge der Brillenlehre für Augenärzte.** By A. BRÜCKNER, Basel. 1st Volume—Glasses and the Eye at Rest. 83 illustrations and 159 pages. Published by Julius Springer, Berlin, 1924.

In the prologue, the author emphasizes the statement that he is endeavoring to present a bridge between the usual ignorance of modern dioptrics on the part of the average oculist, and the highly complex, purely mathematical concepts of Henker, von Rohr, and Gullstrand. To understand this "simple" bridge requires a thorough knowledge of mathematics, up to and including calculus. The first 44 pages are devoted to the laws and mathematics of the general dioptrics so necessary to "Brillenlehre." This is well done, but seems somewhat unnecessarily complex for the average oculist. The second portion, comprising 80 pages, deals with the mathematical relations of the eye at rest and the glasses necessary

for the proper correction of the various ametropias. In this is necessarily included the mathematical constants of the resting eye, and for these, he has adopted in toto the Gullstrand figures. In fact, scarcely a page occurs whereon Gullstrand's name is not mentioned at least once. The third portion consists entirely of mathematical examples and their solution. It is extremely questionable in the mind of the reviewer whether there is the desire or even the need on the part of the average oculist for the theoretical and mathematical knowledge herein contained, although it is served in a fairly palatable form. The printing and the illustrations are good and it is presumed that the mathematics have been checked over by von Rohr so that the accuracy need not be questioned. A fierce Germanism predominates the diction so that extra-Teutonic words are noticeable by their absence.

H. S. G.